

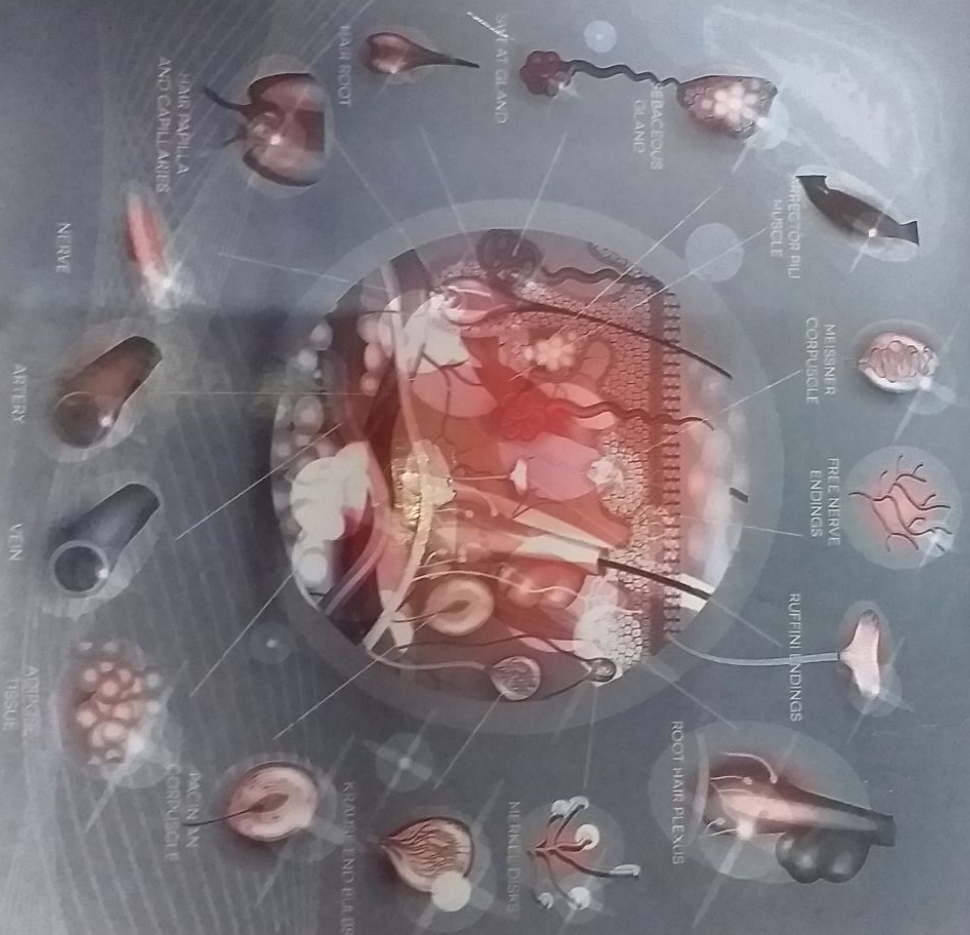


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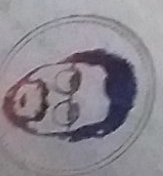
CRASH DERMMA BOOK

FOR UNDERGRADUATES



ABDELRAHMAN REDA

WUDataBot



REDA COURSE

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candidiasis

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Allergic Skin Diseases.....

- Subtypes
- Eczema (Endogenous – Exogenous)
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Skin Anatomy and Functions

CONTENTS LIST

skin anatomy and functions

Scheme

Functions

Primary lesions

Secondary lesions



SCAN TO WATCH



CHAPTER (1)

SKIN ANATOMY AND FUNCTIONS

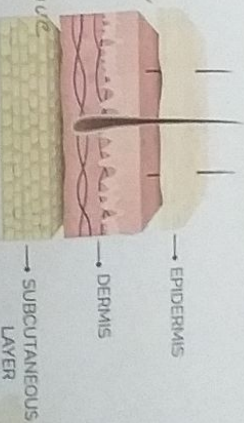
Three Main Layers (3):

1. Epidermis ✓
2. dermis ✓
3. subcutaneous fat ✓

skin appendages (3):

1. hair follicles. *competes* hair shaft → consists of dead, terminally differentiated keratinocytes = nail plates
2. sebaceous glands
3. sweat glands; eccrine and apocrine
4. nails → nail plate → full keratinized, by germinal ridge
→ nail bed → epithelium of nail matrix
→ nail fold
→ nail matrix
* nail growth rate → fingernails → 3mm / month
→ toenails → 1mm / month.

Three Main Layers of the Skin



Epidermis → separated by a basement membrane.

principle cell, keratinocyte

95%

5%

Dermis (15-40) Times thicker than epidermis.
Connective tissue - collagen-elastic tissue-reticular fibers, cells (most cells, fibroblasts, mastocytes).

Subcutaneous Fat

Name	Basal cell layer	Spinous cell layer	Granular layer	Horny layer
Specific	Above basement .M	Spiky cells * have spines	Cells with keratohyalin granules	cells without nuclei - organelles * contain only keratin
Num. of layers	Single layer	5-7 rows	2-3 rows	Many layers

glycolysis in

* epidermis cells renew and turnover every 40 days from basal 2 horny layer.

1-Langerhans cells: (APCs)

2-Melanocyte cells: (APCs)

3-Merkel's cell.

Name	1-Papillary layer	2-Reticular layer
Site	Thin upper layer	Thicker lower layer

Support for: (blood vessels-lymphatic vessels - nerve supply - skin appendages)

*pathological terms:-

- 1-hyperkeratosis → ↑ thickening of horny layer
- 2-parakeratosis → immature keratinization → mede
- 3-acanthosis → ↑ thickening of spinous layer
- 4-acantholysis → loss of coherence
- 1) epidermal & epith. cells
- 5-spongiosis → intercellular oedema in the spinous cell layer

SCHEME

- Shape, dry or 2ry ✓
- Color ✓
- Site ✓
- Itchy - painful - burning ✓
- Associated ✓
 - Systemic disease e.g. viral & diabetes
 - Dermatological (Mucous Membrane - Nail - Hair)
- Healing (with or without) scar ✓
- Complications ✓

FUNCTIONS

*Principles of diagnosis:-

- Personal history
- Complaint
- Present history
- duration
- onset
- site
- symptoms
- acute, rapid, gradual
- relapsing
- chronic

- Examination
 - ↳ distribution
 - ↳ configuration
 - ↳ morphology

- Family history
- med. diseases
- gen. symptoms
- social history
- drug intake
- allergy

investigation

Follow up history

Prevention

Temperature regulation

- Vasoconstriction or dilatation of BVS
- Evaporation of sweat ✓

Protection against UV damage

By Melanin

Immune defense

- stratum corneum integrity.
- Acid pH of sweat.
- sebaceous secretions.
- Antimicrobial peptides

Synthesis

vitamin D from ergosterol by UV

Sensation

- heat, pain
- pressure, vibration, and touch
- Autonomic nerves

Aesthetic

water loss

PRIMARY LESIONS

color

Macule

1 CM patch

(Flat discoloration. There is no elevation, depression)

- Macule
Less than 1 cm in diameter.

- Patch
More than 1 cm in diameter.

Elevation

Fluid

Clear

0.5 CM vesicle
Bulla

Others, pus

- Vesicle Clear fluid collection less 0.5 cm

- Bulla Clear fluid collection more than 0.5 cm

- Pustule pus collection

- 1ry: pus from the start
- 2ry: vesicle that was 2ry infected.

Solid

papule

Nodule

(A circumscribed, elevated, solid lesion)

- Papule less 0.5cm

Surface, flat, conical, round or umbilicated

Papules → confluent → plaques

- Nodule more 1cm.

4 Specific

- Wheal (hive) → Urticaria

C/P: Edematous itchy plaque.
DT: Edema in dermis, sudden

- Burrow → Scabies (mites) (white)

C/P: An elevated channel
Superficial epidermis

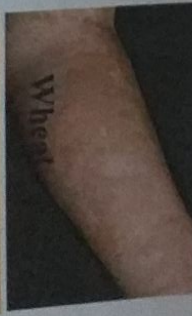
- Comedone → Acne

C/P: Blackheads or whiteheads
DT: Dilated pilosebaceous duct filled with sebum and hyperkeratosis

- Target (iris) → Erythema

Multiform

C/P: Circular erythematous lesions with a vesicle or papule in the center.



SECONDARY LESIONS

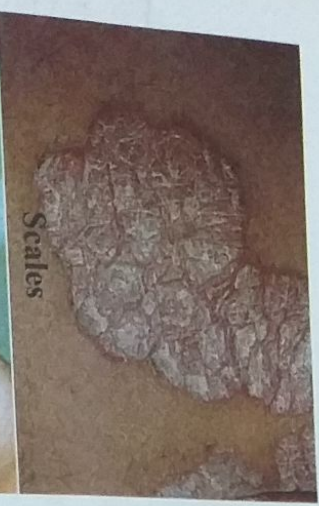
Altered keratinization → in epidermis only
 Bulla
 [ulcer, erosion] → in epidermis only
 ↓
 fissure, crack, fissure

- **Scales** Excess horny layers DT abnormal keratinization
- **Crust** a collection of dried secretions. + coagulated fluids
- **Erosion** a focal loss of epidermis.
 - No penetration below the dermo-epidermal junction
 - heal without scarring.
- **Ulcer** a focal loss of epidermis and dermis.
 - heal with scarring.
- **Fissure** a linear loss of epidermis and dermis

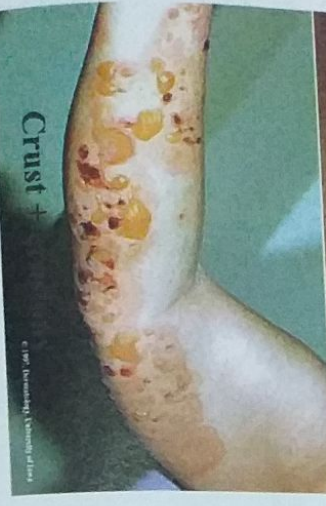
only description
 ↓
 not diagnosis

- **Atrophy** Thinning (epidermis or dermis) → depression in the skin.
- **Scar** Dermal damage (injury or surgery) → abnormal formation of connective tissue → fibrous tissue
- initially thick and pink → white and atrophic.
- **Lichenification** prolonged and repeated irritation → (Thickened+ hyperpigmented)

تقرح



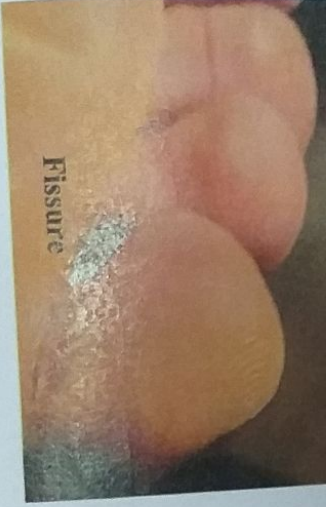
Scales



Crust



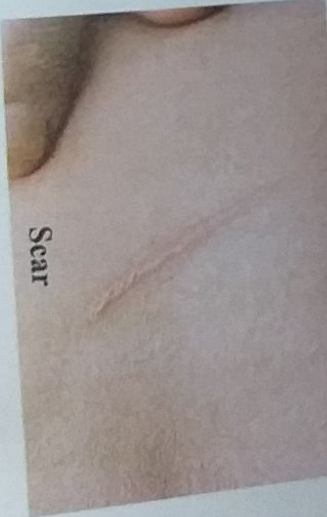
Ulcer



Fissure



Atrophy



Scar



Lichenification

2

Bacterial Infections of The Skin

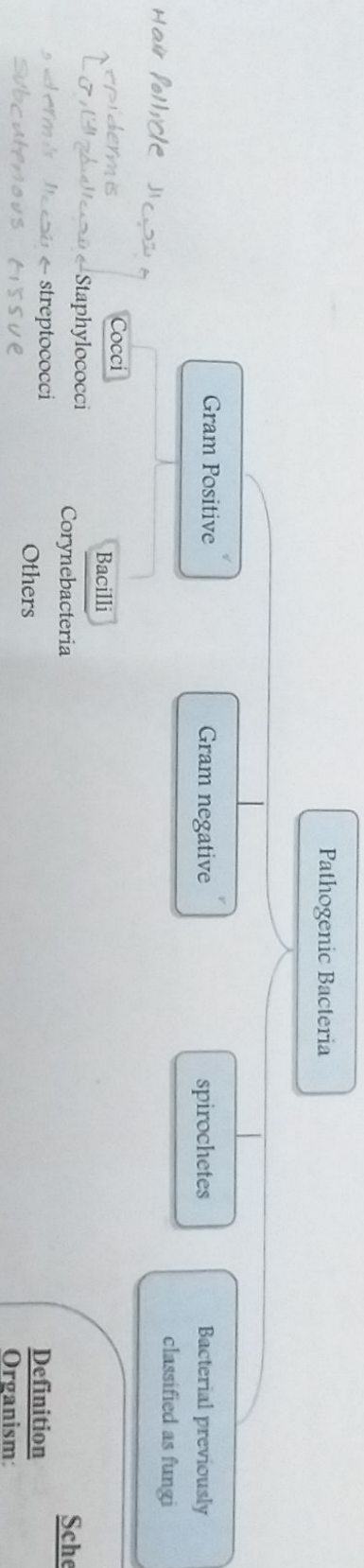
CONTENTS LIST

- General Scheme
- Cutaneous infections (Gram +ve cocci)
- Hair infections
- Nail fold Infection. (PARONYCHIA)
- Mucous Membrane infection. (ANGULAR CHEILITIS)
- Cutaneous gram + bacilli. (ERYTHRASMA)
- Intertrigo



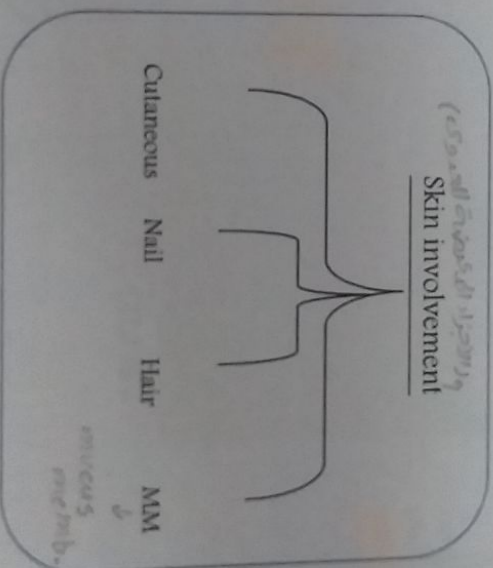
CHAPTER 2

BACTERIAL INFECTIONS OF THE SKIN



The factors involved in this local resistance are:

1. Cutaneous flora → (bacterial interference) *through FFA that destroys pathogens*
2. Dryness of the skin → limits multiplication of bacteria *less bacteria*
3. The constant shedding of the horny layer *desquamation*
4. Sebaceous secretion → natural antibacterial *Sebaceous glands*
5. The low pH of the skin environment (pH 5.5)
6. Antimicrobial peptides → FFA



Normal flora	Pathogenic
Commensals	Harmful
Harmless	

Handwritten note: Skin flora is always good

Definition

Organism:

Clinical

Shape

Color

Site

Itchy / painful / burn

Asso(PDF - Sys)

Complication

Healing

Investigation:

clinical & Culture and swab

Treatment:

- Treat cause
- K-permanganate + Topical AB
- Sys AB
- Surgical incision if abscess

Handwritten notes:
 predisposed factors
 not wound
 DM
 Good hygiene
 prevent - clean

CUTANEOUS INFECTIONS (GRAM +VE COCCI)

Circinate Impetigo

- Single lesions extend peripherally, with central healing → annular, circinate
- Multiple lesions arranged in a circular fashion

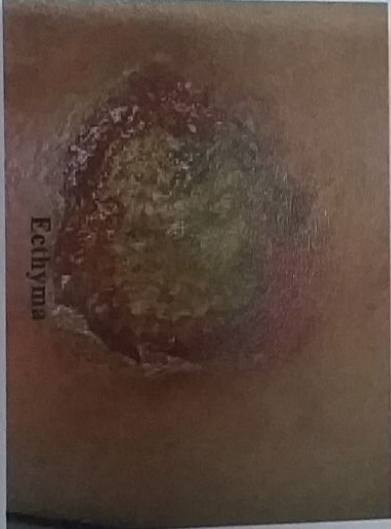
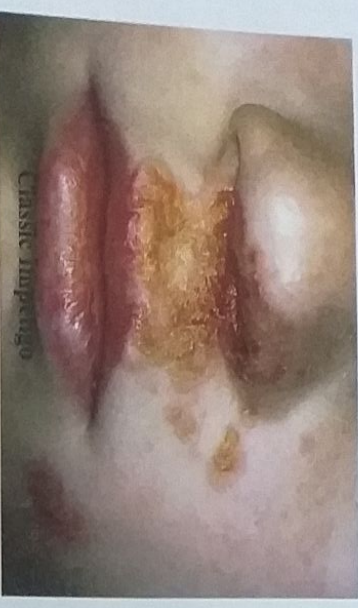
most common bacterial infection of children			
CLASSIC IMPETIGO	Bullous impetigo	ECTHYMA	ERYSIPELAS
CAUSTIVE ORGANISM	Staph aureus or Strept pyogenes	Streptococci	Streptococci
DEFINITION	pyogenic infection of the skin	Acute deep infection	infection of the dermis
SHAPE	1ry: thin vesicle → rupture → 2ry: erosions + crustation	1ry: thin vesicle Rupture → 2ry: thin flat crust (circinate lesion)	well-demarcated red area + hotness, swelling (may have vesicles, pustules or bullae)
COLOR	Dries honey-colored (yellowish-brown) crust	Clear turbid fluid → thin crust	III-defined red area + hotness, swelling
SITE	Around the mouth & nose and the extremities.	Trunk → may be generalized, including palms & soles	Leg Face abdominal wall
ITCHY / PAINFUL	Painful + Itchy	Painful + Itchy	Painful
PREDISPOSING FACTORS	- bad hygiene - overcrowding - malnutrition - pediculosis or scabies.	Neonates & infants (kidneys cannot excrete this toxin)	- Old - Immunosuppressed - Old - Immunosuppressed - Old - Immunosuppressed - Old - Immunosuppressed
COMPLICATION	post-streptococcal glomerulonephritis (rare)	No local lymphadenopathy	Local lymphadenopathy
HEALING	No scarring (Normal Skin)	No scarring	No scarring

DIAGNOSIS Clinical presentation Culture and sensitivity The same The same

Treatment of predisposing factors (Pediculosis and scabies) Topical: (K permanganate - olive oil - topical AB) - Hospitalization - Rest and antipyretics. Topical: AB

HEALING	No scarring (Normal Skin)	No scarring	Scarring	No scarring	No scarring
	✓	✓			

DIAGNOSIS	Clinical presentation Culture and sensitivity	The same	The same	The same	The same
TREATMENT	1. Treatment of predisposing factors (Pediculosis and scabies) ✓ 2. Topical: (K permanganate - olive oil - topical AB) ✓ 3. Systemic AB: (7 days) β-lactamase resistant, cephalosporin. Erythromycin, azithromycin. (1st generation) if patient sensitive (clotaxillin) cephalosporin				



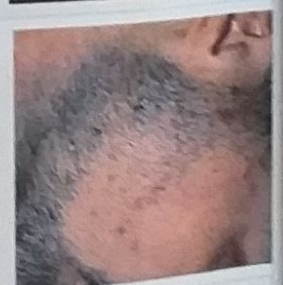
*antibiotics that cover → strept →

recommend in cellulitis eg

- cephalexin
- dicloxacillin
- clindamycin
- combination

HAIR INFECTIONS

		Acute superficial folliculitis (Bockhart's impetigo)		Furunculosis boils		Carbuncle Hair follicles necrosis		Sycosis barbae (الدمى)		Acne keloidalis (folliculitis keloidalis) (الكلاي)	
Definition + organism	Acute bacterial infection of <u>ostium</u> of <u>Hair Follicles</u> by <u>Staph aureus</u>	Acute Deeper bacterial infection of single <u>Hair Follicle</u> by <u>Staph aureus</u>	Acute Deeper bacterial infection of grouped <u>Hair Follicle</u> by <u>Staph aureus</u>	Chronic Deeper bacterial infection of grouped <u>HF</u> by <u>Staph aureus</u>	Recurrent discrete inflammatory papules or pustules → plaques	1ry: small pustules → firm follicular papules → <u>keloid-like plaques</u>	Skin colored → erythematous	Back of the neck (occipital)			
Shape	1ry: <u>pustules</u> → thin walled yellowish-white domed shaped	- Tender hard single or multiple (nodule) → central suppurative and → <u>pus</u> by <u>multiple openings</u> formation of the core (protrusion)	- Tender, indurated (mass) → expulsion → necrotic material + <u>pus</u> by <u>multiple openings</u>								
Color		Erythematous	Erythematous								
Site	At the <u>follicular orifices</u> ? the scalp & face + extremities	Face & neck, buttocks + extremities	- back of the neck (M/C) - shoulders-thigh-buttocks	Beard area							
Itchy / painful	↖ Painful	↖ painful	↖ painful								
Asso. PDF	Occlusion-Maceration – hyper hydration – shaving, plucking hair – Hot weather- Atopic Dermatitis	Old – diabetic – immunocompromised									
Systemic		Bacteremia – Septicemia – Toxemia									
Complication	↖ No	If discharge → may heal with scar									
Healing	↖ No scar	Cause + Culture		Cause + Culture							
Investigation	↖ No	- Topical AB + warm compresses → maturation, drainage and resolution of abscess		- Topical AB							
Treatment	- Topical antiseptics. - Topical antibiotics	- Systemic antibiotics. → <u>oral B-lactamase, penicillin</u>		- Sys AB (resistant Case)							
		- Incision and drainage		- Systemic Steroid if needed.							
		→ <u>mupirocin</u> , <u>sodium fusidate</u>		- Scar → topical & intralesional Steroid							
				Keloid							
				Keloid							



Angioedema:-

- swelling under the skin
- site:- face, throat, larynx, uvula, extremities.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of erysipelas

1. Cellulitis.
2. Angioedema.
3. Acute eczema (atopic dermatitis)

eg

Differential Diagnosis of Syphilis barbae

Pseudo folliculitis:

DEF: non-infectious inflammatory condition

Etiology: (F B reaction to hair) ingrowing hairs in people who shave

Affect: men (with curly hair) (ingrowing hair)

C/P: Papules and pustules on the anterolateral of the neck

Tinea barbae:

DEF: fungal inflammation.

C/P: papules and pustules on beard area with crusting and loosened hair

* differential diagnosis of erythrasma:

- 1- Pityriasis versicolor → site:- upper trunk wood light:- yellow fluorescence. scraping & M.E → hyphae & spores.
- 2- Tinea cruris → active raised border
- 3- Tinea pedis → wood's light.

NAIL INF. (PARONYCHIA)
INFLAMMATION OF THE NAIL FOLD

MM INF. (ANGULAR CHEILITIS)
INFLAMMATION OF THE MOUTH

CUTANEOUS GRAM + BACILLI
CORYNEBACTERIA

Acute Paronychia
Staphylococcus aureus

Chronic Paronychia
chronic: irritants Bacterial + Fungal
[*Candida*]

ANGULAR CHEILITIS

ERYTHRASMA

Definition + organism
Acute Bacterial infection in nail folds by *Staphy + Strept*

chronic: irritants Bacterial + Fungal
[*Candida*]

Chronic streptococcal infection - *Candida* - Riboflavin deficiency (Vit B2)

chronic, localized, superficial infection by *Corynebacterium minutissimum*

Shape Color Site
Hot erythematous Swollen Nail Fold → drain pus

Single or multiple erythematous Swollen Nail Folds: Cuticle: Absent → damage nail matrix

Fissures or cracks or erosions of mouth angles

1ry: Well-defined reddish-brown patches with No active edge. covered with fine scales affect the intertriginous areas

pain
Painful

Recurrent pain

Painful.

Usually non-itchy.

Predisposing factors
Minor trauma usually in Childs

Adult women
Food handlers
Onychomycosis → inflammation nail

Excessive salivation
Ill-fitting dentures
Debilitating diseases + old age

obesity - D.M.
warm humid climate.
adults more than children

Complication

Minor trauma usually in Childs
ES inf. in digits

Onychomycosis → inflammation nail

Debilitating diseases + old age

adults more than children

Healing

Investigation

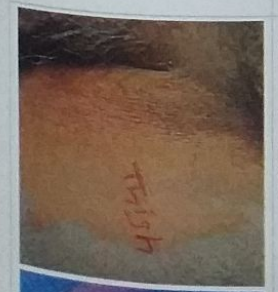
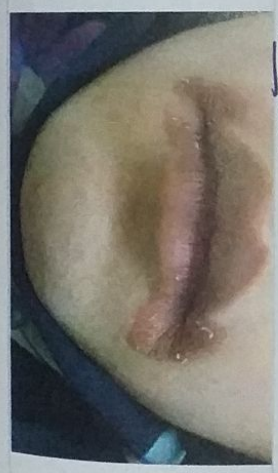
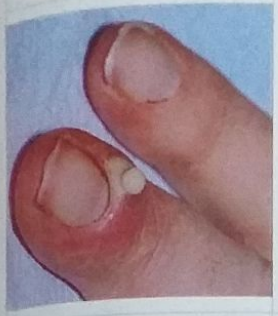
Treatment

- Drainage of pus.
- Topical & Sys AB

- Avoid: water and chemical
- Topical AB & Steroid. → anti-inflammation
- sys. Antifungal.

- TTT Predisposing factors + Vit B complex
- Topical antifungal &/or topical antibiotic, Steroid

- white field
- azole
- Topical antifungal + topical antibiotic
- Systemic erythromycin (if resistant or disseminated) for tetra cycline.
- antibacterial soap to prevent relapse.



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children.

INTERTRIGO

intertriginous areas

- Definition: inflammation of skin areas in between folds
- Site: behind the ears, under the breasts, axillae, groins and skin folds in obese

- Causes of intertrigo: → obvious friction & irritation in moist

1. Simple intertrigo due to friction. → GP A
2. Bacterial: Erythrasma + Streptococcal intertrigo →
Longitudinal painful fissure of the skin folds.
3. Fungal: Tinea cruris + Candidal intertrigo.
4. Flexural psoriasis.
5. Seborrheic dermatitis.
6. Contact Dermatitis

- Treatment of intertrigo

According to etiology

- In case of bacterial

- potassium permanganate lotion 1/8000.
- Topical AB
- Systemic antibiotics

affected { children
infants.
obese adults.



Candidal intertrigo

DD:-

- erythrasma
- dermatophyte infe.
- IgSI → intertrigo.

*Clinic. Pic. :-

- Sharply demarcated, intensely erythematous patches or thin plaques.

3

Viral Infections of the Skin

CONTENTS LIST

General Scheme

HERPES SIMPLEX

VARICELLA (CHICKENPOX)

Warts

MOLLUSCUM CONTAGIOSUM

You TubeTM
SCAN TO WATCH



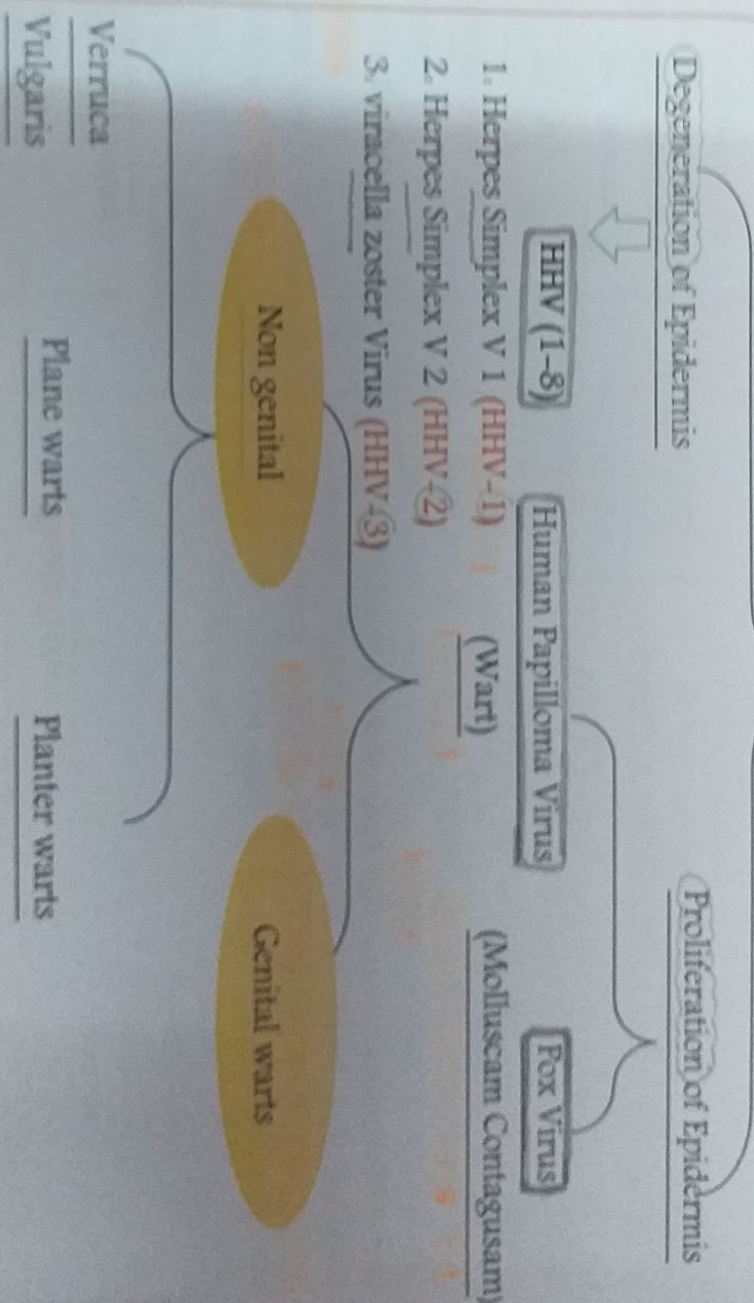
CHAPTER 3

VIRAL INFECTIONS OF THE SKIN

Scheme

- Ore + IP *infection, per-oral*
- Mode of Transmission:
- Clinical
 - Shape
 - Color
 - Size
 - Itchy / painful / burn
 - Assoc (FDF - Sys)
 - Complication
 - Healing
- Investigation
- TTT Acyclovir or destruction

Viral Infection of Epidermal Keratinocytes



HERPES SIMPLEX

Organism: two types of herpes simplex virus:

a- Type I: facial

→ genital also cause

b- Type II: genital

→ facial also cause

(any type of herpes simplex virus can be considered a causative agent for oral & genital herpes)

Pathogenesis:

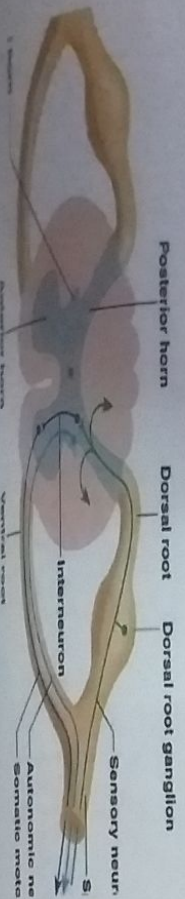
1st attack: multiplication (disseminated lesions) → systemic manifestations (fever – generalized LN) → Latent in dorsal root ganglion (1st lesions usually are subclinical)

2nd attack: stress → + virus in dorsal root ganglion → localized lesions → no systemic manifestations

Herpes Simplex V Type (1)

Herpes Simplex V Type (2)

1 st attack	2 nd attack	1 st attack	2 nd attack
✓ Systemic Manifestations	✓ No Systemic Manifestations	✓ Systemic Manifestations	No Systemic Manifestations
✓ Disseminated lesions	✓ Localized lesions	Disseminated lesions	Localized lesions



VARICELLA (CHICKENPOX)

Organism: varicella-zoster virus.

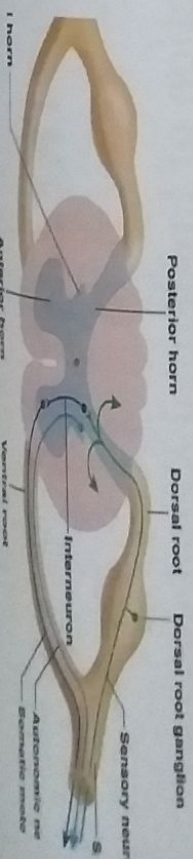
a- Varicella, Chicken Pox

b- Zoster, Herpes Zoster

Pathogenesis:

1st attack (chicken Pox): replication in Mucous membrane → blood → viremia (systemic) → cutaneous eruptions (vesicles mostly trunk) → latent in dorsal root ganglion (mostly thoracic) / cervical / trigeminal (ophthalmic)

2nd attack (herpes Zoster): stress → + virus in dorsal root ganglion → localized lesions (unilateral dermatome) → no systemic manifestations.



Types

(disseminated) Type 1

(localized) Type 2

Recurrent

Varicella (CHICKENPOX)

HERPES ZOSTER

H8 3

Organism

HERPES SIMPLEX

Varicella-zoster virus

Types	Type 1	Type 2	VARICELLA (CHICKENPOX)	HERPES ZOSTER
-------	--------	--------	------------------------	---------------

IP	(Disseminated) Type 1 Iry attack Herpetic gingivostomatitis	(Localized) Type 2 Recurrent attack Primary attack Up to week	Primary attack 2-3 weeks	Secondary attack Recurrent
----	--	--	-----------------------------	-------------------------------

MOT	Oral Contact direct contact	Sexual Contact	Droplet infection. Direct contact.	multiple vesicles surrounded by erythematous base.
-----	--------------------------------	----------------	---------------------------------------	--

Shape	(recurrent attack of genital herpes presented by recurrent painful erosions)	(Vesicles) → erosion	Iry lesions → vesicles (macules, papules, vesicles, pustules crusts)	
-------	--	----------------------	---	--

Color		On erythematous base		
-------	--	----------------------	--	--

Site	Disseminated (gingivostomatitis)	Grouped (around mouth)	- Females vulva, vagina and cervix. - Males Glans, shaft of the penis and scrotum.	(grouped vesicles unilateral) (2) in distribution - If Bilateral → in cases of Immunocompromised (HIV-Malignancy-Steroid)
------	-------------------------------------	---------------------------	---	---

Burning sensation	↑ 2-3 days 1st all	Burning	Itchy + Burning	Burning (mild-severe)
-------------------	--------------------	---------	-----------------	-----------------------

Systemic manifestations	✓ fever & local lymphadenopathy	No sys. manifestations	✓ fever & local lymphadenopathy	Prodomal stage: 1-2 days → (FAHM) + urticaria DD → papular urticaria + scabies
-------------------------	---------------------------------	------------------------	---------------------------------	--

Predisposing factors		- Fever-Stress - anxiety - Flu - Exposure to sun. - Trauma - menstruation	* sexual contact	- Immuno.C - Stress - Malignancy - Steroid - Unknown
----------------------	--	--	------------------	--

Complications	encephalitis	(Rare)	* in pregnancy → neonatal herpes	Rare
---------------	--------------	--------	----------------------------------	------

Healing		Crustation → no scar		Post-inflammatory hyperpigmentation + scars → may be absent
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Investigations	clinical / No	Sexual partner should be examined	No	No
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* To prevent neonatal

Topical:

Antiseptics: Gentian violet 2%, potassium permanganate solution.

Systemic:

Acyclovir 200 mg 5 times/day for 7-10 days

Treatment

If 1st attack Herpes proterialis few days before delivery → cesarean section



Topical

Antiseptics e.g. gentian violet paint or potassium permanganate solution.

Systemic (within 72 hr)
Antiviral → Acyclovir

800mg/5times / 5day

In case of Children:
(severely ill or immune-compromised) 20 mg/kg max 800 mg per dose 4 times / 5 days.

Systemic (within 72 hr)

Antiviral → Acyclovir 800

mg / 5 times / 7-10 days

Painkillers are indicated whenever there is pain.

Common sites of

herpes simplex type 1:

- Herpetic gingivostomatitis.
- Herpetic keratoconjunctivitis.
- Herpetic whitlow (fingers)
- Herpetic affection as in dentists

2nd children }
Oral herpes

Common sites of herpes zoster

- Thoracic affection.
- Cervical affection (usually C 2, 3, 4)
- Trigeminal affection (including ophthalmic division)
- Lumbosacral affection

Complications of Herpes zoster:

- Post-herpetic neuralgia (persistence of pain after healing of cutaneous lesions).
- Secondary infection. → pustule → scar.
- Scarring of the skin.
- Gangrene of the skin.
- Eye: (herpes zoster ophthalmicus).
- Affection of the geniculate ganglion (Ramsay Hunt syndrome).

Keratinocyte proliferation and tumor formation

Warts

rough surface

Genital warts

Keratinocyte Proliferation and Tumor Formation

Warts

Organism	through surface Verruca vulgaris → Ordinary warts most common	Plane warts	Plantar warts	Genital warts (condyloma acuminata)
IP				
MOT	Direct contact Autoinoculation	Human papilloma viruses 1-8 months (up to years)	← (friend of life)	Sexual contact
Shape	(Papule), verrucous (irregular) wart = rough surface papules	Flat smooth surface (papules)	Endophytic (Papules) ↳ = in world (invagination)	Multiple pedunculated cauliflower (papules) & masses (rough surface)
Color		(Grayish)		
Site	- Face - Extensor surface of hands: mostly children		soles or in between the toes (against bony prominence) ↳ (usually) painful	- Female: vulva, vagina and CX - Men: glans and penile shaft.
Itchy/burn/painful	Asymptomatic		Painful (usually)	Asymptomatic
Systemic manifestations/pdf	—			Females :- - Huge cervical lesions → - Obstructive normal delivery → - Cesarean section. - CIN - Cervical Cancer
Complications	—			

Healing

According to destructive method
Hypo/hyperpigmentation

Differential Diagnosis

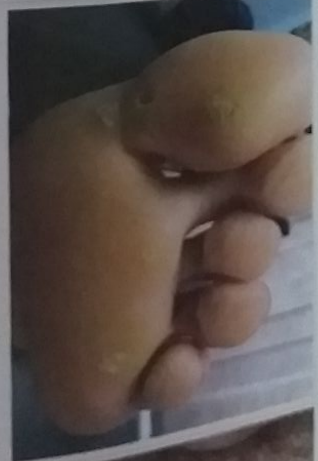
- Callus:**
- Hyperkeratotic area
 - Site: weight bearing area
 - Specific: crossed by skin creases

Condyloma lata of syphilis

Destruction

Non genital				
Topical	Chemical	Thermal	Electric	laser
Topical keratolytic	- acetic acid. - salicylic acid 40 %: Planter warts (or Mix)	Cryotherapy → liquid nitrogen (-196°C) → X in cage of facial lesions as it leads to hyper pigmentation	Electric cautery	CO ₂
Genital				
+ Podophyllin 25% in tincture benzoin (contra-indicated in pregnancy) - trichloro acetic acid (TCA) 70-90% (safe in pregnancy)				

Treatment
(معالجه)
(may resolve spontaneously without treatment)



(POX VIRUS)

• Organism: POX VIRUS

@AUDatabot

MOLLUSCUM CONTAGIOSUM (POX VIRUS)

- Organism: POX VIRUS
- Incubation periods: 6-9 months
- Mode of Transmission: direct contact (Highly contagious lesions) – less indirect. – highly infectious
- Shape + color: (single or multiples) shiny pearly white umbilicated papule in the center
- Site: Face, genitalia, trunk.
- Painless x
- Systemic manifestations / predisposing factors: NO x
- Complications: NO x
- Healing: Without Scars x
- Treatment:
 - Mechanical methods: squeezing papule → Expressing the content by (forceps or curettage) → cauterizing the base. (The best)
- ✓ - Any Destructive Methods; phenol – electric – cryotherapy – Laser.



4

MYCOBACTERIAL INFECTIONS

CONTENTS LIST

General Scheme

Mycobact. Tuberculosis. (route – immunity – clinical picture)

Leprosy. (Hansen disease)



SCAN TO WATCH



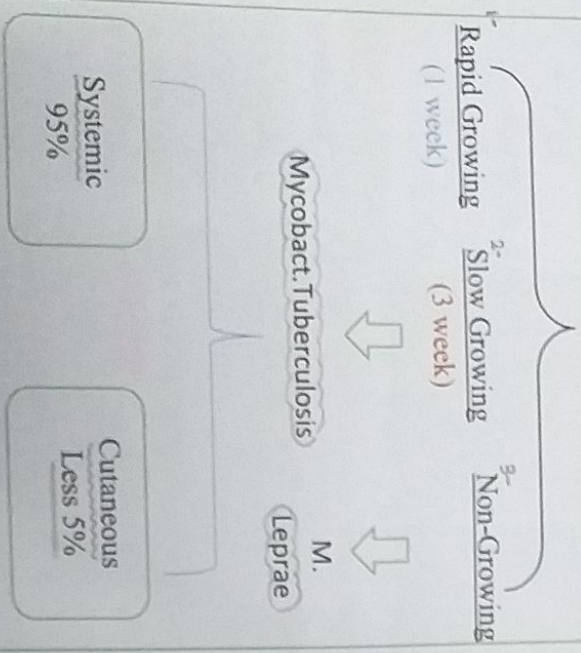
→ true T.B. skin:- invasion of skin by tubercle bacilli

→ tuberculids:- allergic response of the skin to remote tubercle bacilli or their toxin

MYCOBACTERIAL INFECTIONS

1. Slender - Non-profile
2. Acid-fast (once stained) → resist decolorization
3. Aerobic

Classification according to culture



Cutaneous Tuberculosis Clinical Presentations depend on

1. Route of skin involvement.
2. Immunity & resistance of the patient
3. Virulence + Number

Scheme

- Def:
- Org + IP:
- Route of Transmission:
- Immunity:
- (Clinical)
 - Shape: small pap > nodules
 - Color: red to brown
 - Site → according to penetration site.
 - Itchy / painful / burn
 - Asso (PDF - Sys)
 - Complication: long duration > destruction
 - (Healing) scar
- Diagnosis:
- TTT:

Diagnosis (SCBPT)

1. Swab: Ziehl-Neelsen stain
2. Culture: Lowenstein-Jensen medium (3 weeks)
3. Biopsy: caseating granuloma / necrosis
4. PCR: DNA Detection
5. Tuberculin Test: show past immunity (2)
 - Positive → (past exposure + good immunity)
 - ① Infection
 - ② Vaccination
 - Negative → (No past exposure or low imm)

Treatment

- General measures
 - Antituberculous drugs:
 1. Isoniazid → S/E: Polyneuropathy [NM]
 2. Rifampicin → S/E: hepatotoxic + Red urine
 3. Pyrazinamide → S/E: hepatitis + Arthritis
 4. Ethambutol → S/E: visual disturbance
 5. Streptomycin → S/E: ototoxicity
- 4x2 + 2x4
 1 Drug for 2 in then 2 Drugs + 4 m
 All drugs are taken on empty stomach.
 Small lesions can be excised.

PTB developed in the lung
[Subpleural 1st (exsion)]

Route Of Infection And Immunity

Route is

(for cutaneous) Exogenous

(direct inoculation)

(cutaneous) Endogenous

(spread) Hematological

Disease	Immunity	Tuberculin test	Incub. Period	Clinically	Diagnosis	TTT
Chancres Tuberculosis Chancre	No past immunity	-ve Tuberculin	1 Month	Single Red to brown Papules to nodules → ulcer at site of trauma Painless	No Association	Healed with Scar (exsion)
TB Verrucosa Cutis	Good Past immunity (Vaccine-Infection)	+ve Tuberculin	1-2 Months	Same+ wart like plaque Site: Extremities (trauma) → greyish with redness Viral warts		Healed with Scar
Scrofuloderma		Weak +ve Tuberculin		Red to brown Papules to nodules → ulcer → Sinus site: extension direct from bone - joints - LN Epididymis		Healed with Scar
Orificial T.B	Low immunity	-ve Tuberculin		Red to brown Papules to nodules → ulcer → Sinus site: periorificial → extension from internal organs to its orifices. → sys. association → painful		Healed with Scar
Acute Miliary Tuberculosis		+ve Tuberculin		Red to brown Tiny papules with bad general condition site: trunk		Healed with Scar



Lupus Vulgaris (M/C)

- Org (Routes 4/11-1/14)
- Route Of Transmission: external – internal - hematological
- Immunity: good past immunity
- Tuberculin test: +ve → moderate to high immunity
- Clinical site :- Face, limb, buttocks
 - Shape + color: red to brown small pap → nodules (apple jelly nodule) → plaque → form scar contractile unhealthy scar
 - Site: face – cheeks - nose
 - painless
 - Complication: long duration > destruction of cartilage
- Healing: scar → thick, contractile and unhealthy (contains active nodules).
- Diagnosis: SCBPT + Diascopy + mantoux test
- TTT: Mentioned B4



Diascopy test

Pressing on the lesion by a glass slide leads to fading of the erythema and the nodules appear as yellow-brown spots

erythema leads to fading of the erythema and the nodules appear as yellow-brown spots

Organism (Prince)

- Obligated Intracellular
Schwan C (macroph cells)
MQC
- Long incubation period
prefer low temperature
year
- Variant Presentation

Leprosy Hansen disease

Diagnosis (SCBL)

1. Smear: (Nose - Slit Skin) modified Ziehl-Neelsen stain (Fite stain)
2. Culture: vivo using the nine-banded armadillo. (not applicable) ✗
3. Biopsy.
4. Lepromin Test: show past immunity
 - Positive → (past exposure + good Imm)
 - Negative → (No past Exposure or low imm)
 } Tuberculin test

Treatment (DCCR)

(Dapsone - Clofazimine - Rifampicin)

- Paucibacillary leprosy (TT, BT): 6 M
 - Dapsone (bacteriostatic) 100 mg/d
 - Rifampicin (bactericidal) 600 mg/m
- Multibacillary leprosy (BB, BL, LL): 2 Y
 - Dapsone 100 mg/d
 - Clofazimine 50 mg/d
 - Rifampicin 600 mg/m
 - Clofazimine 300 mg/m.

Organism: Mycobacterium leprae.

IP: Up to 30 years (3-5) years

MOT: Debate →

- Droplet infection.
- Blood
- Inoculation to Skin or MM

Immunity:

Clinical

- Shape:
- Color:
- Site
- Itchy / painful / burn
- Association (PDF - Sys)
- Complication:
- Healing:

Diagnosis: SCB + Lepromin

TTT:

Tuberculoid leprosy (TT)

Immunity

Good Reaction → localization → Destruction
→ early

Lepromin test

Num: single up to 5. 1-5

Shape: well-defined macules & patches.

Color: hypopigmented + loss of hair.

Site: unilateral – asymmetrical

Sensation → (Early affected)

Impaired Sensory: pain - temperature.

Autonomic: hypohidrotic

nerve destruction

atrophy of interosseous ms.

→ Food drop

*not [itchy - painful - burn]

Clinical picture

Association

Investigations

SCBL

Biopsy:

linear granuloma following a nerve + no bacilli

inflammation around nerve

SCBL

Biopsy:

normal collagen separate Epidermis & Dermis + many bacilli

- Diffuse Foamy histiocytes in dermis: Macrophages

SCBL

Biopsy:

normal collagen separate Epidermis & Dermis + many bacilli

- Diffuse Foamy histiocytes in dermis: Macrophages

SCBL

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Lepromatous leprosy (LL)

Immunity

Weak Reaction → no localization → late onset manifestation

Lepromin test

Num: multiple.

Shape: ill-defined macules & papules & nodules

Color: skin colored

Site: bilateral – symmetrical as there is localization

Sensation →

Early: intact sensation → (المشعر في وقت مبكر)

1. Gloves & stocks loss sensation

2. Loss of the outer 1/3 of eye brows

3. Leonine facies (شبه وجه الأسد)

can be disseminated to testis – eyes – bone – reticuloendothelial system

involvements:-

mm. - eyes - testis

SCBL

Biopsy:

normal collagen separate Epidermis & Dermis + many bacilli

- Diffuse Foamy histiocytes in dermis: Macrophages

SCBL

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SCBL

Biopsy:

normal collagen separate Epidermis & Dermis + many bacilli

Borderline leprosy (BL)

Immunity

In-between

Lepromin test

Num: multiple.

Shape: ill-defined macules & papules & nodules

Color: skin colored

Site: bilateral – symmetrical as there is localization

Sensation →

Early: intact sensation → (المشعر في وقت مبكر)

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Immunity

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normal collagen separate Epidermis & Dermis + many bacilli

- Diffuse Foamy histiocytes in dermis: Macrophages

SCBL

Biopsy:

normal collagen separate Epidermis & Dermis + many bacilli

5

PARASITIC INFESTATIONS

CONTENTS LIST

General Scheme

Scabies

PEDICULOSIS



SCAN TO WATCH



CHAPTER 5

PARASITIC INFESTATIONS

Parasitic infections
- Scabies
- Pediculosis

• ordinary scabies

- Org + IP: **Sarcoptes scabiei hominis** - 3-4 weeks
- MOI: from human or animal
- Clinical:

Shape: **Pleomorphic mainly burrows (specific)** (pic B)

Site:

- **Folds** as **interdigital of hands** (pic-A)
- flexor aspects of wrists, elbows, axillary folds
- penis and scrotum, buttocks
- No Face Involvement except infants

Itchy: **Night - all family**

Predisposing factors:

- Overcrowding poor hygiene
- Wars - natural disasters

Complication: **post scabies pruritus - pyogenic**

inf.

DD: itching (insect bites - papular urticaria)

Atopic dermatitis - contact dermatitis

Variants →

- Investigation: clinical & 10% potassium hydroxide?
- TTT: see later

Scabies

Variants

- Age:
 - Ordinary scabies adult
 - Scabies in infants (pic-D): face, palms and soles → **Vesicular lesions**
- No lesions:
 - 1. Animal scabies: No burrows - lesions at contact site - self-limiting.
 - 2. Scabies in (clean) NO burrows - due to frequent bathing.
 - 3. Scabies incognito: reduced signs and symptoms → due to using steroids.
- Severe:
 - 1. **Norwegian scabies (Pic-E)** (millions of mites, **keratotic scabies - not itchy**): Present in immune-compromised, physically disabled and mentally deficient individuals.
- Hypersensitivity:
 - **Nodular scabies (pic.C)** (post scabietic nodules)
 - Persistently pruritic nodules after scabies therapy.
 - Genitalia, crural area and axillae.
 - Topical or intralesional corticosteroids → TTT

A

C

D

E

@AUDataBot

Investigations/ Diagnosis:

10% potassium hydroxide test
scraping a burrow with a blunt scalpel + 10%
potassium hydroxide on a
microscopic slide → mites, eggs or fragment of
egg shells

Treatment

Specific Treatment:

A. Topical application: Topical therapy is given usually overnight.

- Infant from head to toe. Adults, face and neck are spared.
- Sulfur: 5-10% (at night for 3-5 days).
- Permethrin 2.5% for children and 5% for adults (at night, at day 1 & 8).
- Benzyl benzoate emulsion 25-33 %.
- Gamma benzene hexachloride 1% (at night, at day 1 & 8).
- Malathion 0.5%. (at night, at day 1 & 8).
- Crotaniton 10 % (at night, for 3-5 days).

B. Systemic therapy: Ivermectin.

The only topical treatments
used in pregnant females
are sulfur and permethrin

General Measures:

- All members should be treated
- Clothing: boiled or ironed or kept in bag for 10 days.

General Treatment:

- Itching: Oral antihistamines.
- Secondary infection: should be treated at first

with antibiotics
if there is infection

PEDICULOSIS

	Head louse	body or clothing louse Natural habitat is the clothing	Pubic louse
Org + IP	Pediculus Humanus capitis ^{scalp}	pediculus humanus corporis ^{body}	Phthirus pubis ^{pubic area}
MOT	Direct & Indirect	Direct	Physical & Sexual contact
Clinical	Shape: Multiple nits – lice. In the scalp → mostly → occipital and post-auricular. Itchy: severe itching (characteristic manifestation) Predisposing factors: overcrowding. Complication: bacterial infection → (scalp impetigo) → enlarged Cervical lymph nodes	Shape: Multiple scratch marks With hemorrhagic spots all over the body Itchy: severe itching (characteristic manifestation) Predisposing factors: overcrowding. Complication: bacterial infection	Shape: Multiple lice grasping hairs with huge claws- Nits. In Pubic Area – axillary hair, eyebrows, and eyelashes Itchy: severe itching in pubic (characteristic manifestation) Predisposing factors: sexual C. Complication: bacterial infection → red spots in underwear because of its excreta.
Investigation	General Measurement: - Head louse: Removal of eggs with fine toothed comb +/- vinegar. - Pediculus humanus corporis: Clothes Ironing → Kill → lice and eggs. - Phthirus pubis: Shaving the pubic area is the first step in the treatment.	Diagnosed clinically	
TTT	General Treatment - Itching: Anthistaminic - pyogenic: systemic antibiotics at first.		

Specific Treatment

- Anti-pediculous agents (day 1 and 8):
 - Topical
 - Permethrin: 2.5% for children and 5% for adults.
 - Benzyl benzoate emulsion 25% ^{adults}
 - Malathion 0.5% → crab lice ← in 2 weeks
 - Gamma Benzene hexachloride 1%.
 - Systemic
 - Oral Ivermectin.

TTT



6

FUNGAL INFECTIONS OF THE SKIN

CONTENTS LIST

General Scheme

TINEA CAPITIS

Other types of Tinea

PITYRIASIS VERSICOLOR

candidiasis



SCAN TO WATCH

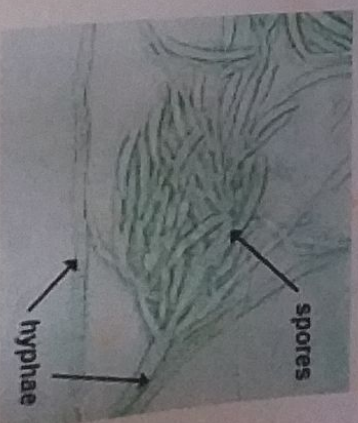
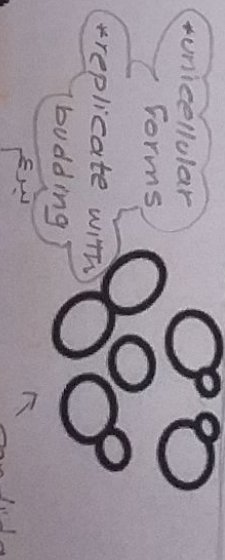
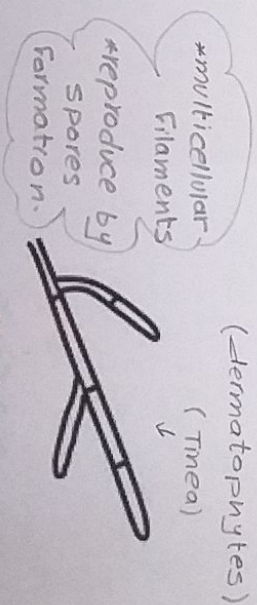
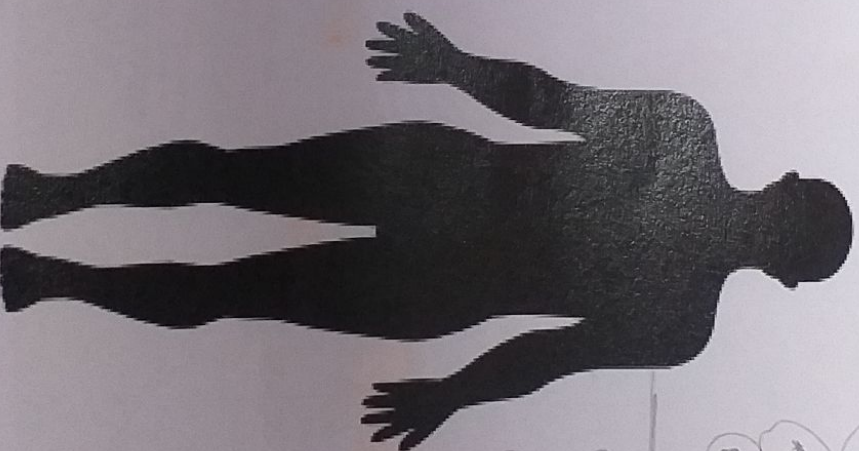


CHAPTER 6 FUNGAL INFECTIONS OF THE SKIN

General scheme

- Org *Healing centre, active edge* *if possible*
- Clinical:
 - Shape: circinate or raised active edge or $\leftarrow$ D
 - satellite lesions $\leftarrow$ D
 - Color:
 - Site
 - Itchy
 - Asso (PDF - Sys)
 - Complication:
 - Healing
 - DD
- Investigation:
 - Wood's light examination
 - Direct microscopy (KOH 10%): Hyphae-Spores
 - Culture: Sabouraud's agar $\rightarrow$ Macroscopic appearance - Microscopic appearance
 - TTT: Avoid humidity + Topical + Sys Antifungal.

الجلد له مركز يشفي
في حال امكانه
في حال وجود
فطريات



→ most of infections:-
 1. direct contact.
 2. indirect contact.
 3. contaminated air.

dermatophytes **TINEA CAPITIS**

RINGWORM OF THE SCALP
 (Healing - LN?)

Why T.C. more common in Childs?
 Adult → more androgen → more sebum
 → fungistatic → less in adult

	Black dot ringworm	Scaly ringworm	Favus	severe inflammatory reaction (animal origin)
Organism	T. violaceum T. tonsurans	T. violaceum Microsporum canis " audouinii	T. schoenleinii	T. verrucosum T. mentagrophytes
Hair & scales: single or multiple scaly patches	Specific → with shortly cut hairs (black stumps) Scaring: NO	Hair & scales: single or multiple scaly patches Specific: More scales Scaring: NO	Hair & scales: Diffuse > entire scalp Specific: hairs surrounded by thick yellow crust → cup shaped (sulfur cups or scutula). Scaring: yes → permanent alopecia with scattered "coconut hairs" in it	Hair & scales: single or multiple (swellings) with - Hair easily detached - few pustules Specific: Painless Scaring: Yes
Clinical	LN: No	LN: No	LN: No	LN: No according to severity of reaction

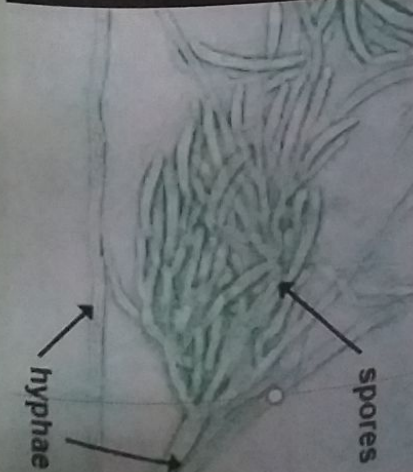
• **Wood's light examination: screening**

- (Microsporum) + audouinii → green fluoresce
- T violaceum → not fluoresce

• **Direct microscopy:** (scrapping + KOH 10% + warming)

- Hyphae
- Spores

• **Culture:** Sabouraud's agar (2-4 weeks)



- **Topical (not effective):** Imidazole group of drugs.
- **Systemic:**

➤ **Griseofulvin**

Absorption: best after fatty meal
 Dose: (12.5 Mg/Kg) (each tab 125mg - Max 6 tablets)

- topical antiseptic in kenon to remove scales.

- **Topical (not effective):** Imidazole group of drugs.

Systemic:

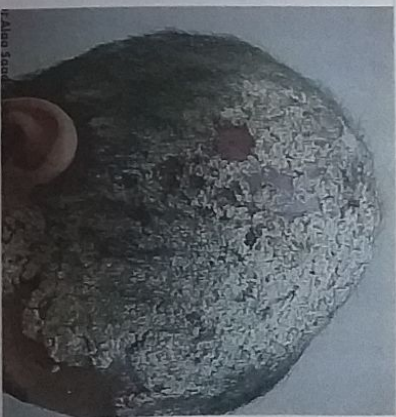
Griseofulvin

جريسوفولفين

- Absorption: best after fatty meal
- Dose: 12.5 Mg/Kg (each tab 125mg - Max 6 tablets)
- Duration: 6-12 weeks.
- S/E: GID, photosensitivity, drug induced SLE.
- Contraindicated: pregnancy - hepatic failure - photosensitivity.
- Not effective in candidiasis and pityriasis versicolor
- Other systemic antifungal: Itraconazole or Terbinafine

- topical antibiotic in kenon to remove scales.

TTT



Differential diagnosis

Scaly scalp

Seborrheic dermatitis
impetigo
psoriasis

Scaly localized non cicatricial alopecia

(مناطق مغطاة بالجلد) Alopecia areata,
(حالات نادرة من الصلع) trichotillomania.

circumscribed vesicular lesion

Other types of Tinea

1. Tinea capitis
2. Tinea pedis

Tinea manuum: Fungal infection of dorsum of the hand

- Common in adult males
- The commonest form

TINEA BARBAE

TINEA CORPORIS

TINEA CRURIS

ONYCHOMYCOSIS

TINEA PEDIS

Site The jaw angle (beard)

Trunk and limbs

upper medial side of the thigh extend to buttocks. (Epidermophyton floccosum + Trichophyton rubrum)

Nail plate

itchy interdigital maceration bet. the 4th / 5th toe-web space

Shape

Similar to kerion (inflammatory - type) less inflammatory type. (tinea corporis)

Complication

Healed with Cicatricial alopecia = scarring

No

DD of intertriginous

- Circinate impetigo
- Pityriasis rosea
- Erythrasma.
- Candidiasis of the groin.
- Intertrigo.
- Psoriasis (flexural).
- Seb. dermatitis

Psoriasis

Caused by:

- Dermatophytes.
- Candida.
- Gram-negative bac.



TTC → Tioconazole nail sol. 28% (21 days)

PITYRIASIS VERSICOLOR

Other cutaneous fungal infections

Chronic

PO = scales = mud color
PITYRIASIS VERSICOLOR
 Pityrosporum ovale → Malassezia

Other cutaneous fungal infections

Oral candidiasis
 (oral thrush)

Genital candidiasis

Candidal intertrigo

Chronic mucocutaneous candidiasis

Candidal paronychia

Org

Malassezia yeasts

Shape: multiple well-defined macules and patches + fine scales.

Color: hyper or hypo-pigmented

Site: trunk and neck, Face in children

Predisposing Factors:

- humid
- oily skin
- pregnancy
- malnutrition
- infection in hairy cell leprosy

Specific:

scrubbed →

Erythematous base

Shape: whitish curd-like patches (pseudomembrane)

Of buccal mucosa and tongue

Shape: whitish patches with erythema in vagina

glans penis

most common

Candida albicans

shape: well defined, erythematous patch in Groin, Axillae

sub mammary.

specific: edge is festooned with satellite papules & pustules

due to: immune def

shape: of yeast

- chronic paronychia

- oral candidiasis

- hyperkeratosis on hands and feet

- subungual hyperkeratosis

- subungual hyperkeratosis

due to: immune def

Site: Nail folds

Predisposing factors: diabetes, obesity, moisture, immunocompromised individuals, prolonged intake of broad-spectrum antibiotics and old individuals

• Wood's light examination: high golden yellow fluorescence

• Scraping & MF → spores & short hyphae not balls in a spore

Investigation

• Wood's light examination

• Direct microscopy (KOH 10%): Yeasts

• Culture: Sabouraud's agar

TTT

• Avoid humidity (Recurrence very high)

• Topical: Sodium thiosulphate + others.

• systemic anti-fungal.

(for tinea and candida)

• Itraconazole

• Avoid humidity

• Topical therapy:

- Broad-spectrum Azole Group e.g. Imidazole group

- Tincture iodine

- Whitfield ointment

- sodium hyposulphite pityriasis versicolor

• systemic anti-fungal.

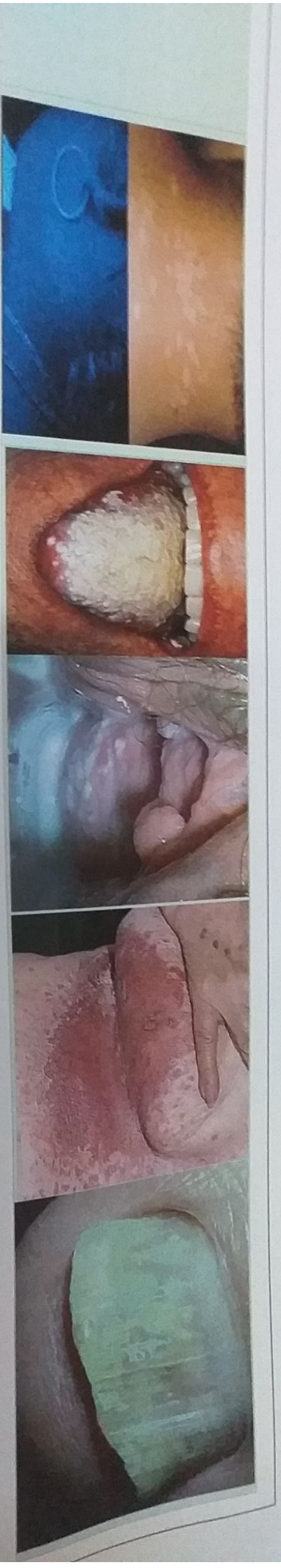
DD of napkin rash

- Contact dermatitis: young infants, ill-defined
- Psoriasis: well-defined border - no active rash - no scale
- Tinea cruris: Raised active edge

Systemic therapy

- Griseofulvin Tinea Capitis. only
- Not Effective (TVC - Candida)
- Terbinafine dermatophytes only.
- Itraconazole, Broad-spectrum

circumscribed vesicular scalp hyperkeratotic type



Tinea incognita

Def: Fungal infection modified in appearance

Shape: less erythematous and the active edge becomes less prominent)

Etiology: due to using topical or systemic steroids.

7

Allergic Skin Diseases

CONTENTS LIST

- Subtypes
- Eczema (Endogenous – Exogenous)
- Urticaria
- Urticarial vasculitis
- Angioedema
- PAPULAR URTICARIA
- ERYTHEMA MULTIFORME
- ERYTHEMA NODOSUM
- Steven's Johnson Syndrome
- Fixed Drug Eruption



SCAN TO WATCH
PART 1



SCAN TO WATCH
PART 2



CHAPTER 7

ALLERGIC SKIN DISEASES

ECZEMA
Non-infective

URTICARIA AND
ANGIOEDEMA

Eruption DT Infection
(Mostly)

Eruption DT Drugs
(Mostly)

Def. :- inflammation non-infectious

Acute stage: (inflammatory stage)

- Ill-defined erythematous patches
- Papulo-vesicular eruption
- Exudation of serum

Subacute stage:

- Erythema and scaling

Chronic stage:

- Dryness and lichenification

Exogenous eczema

Irritant contact dermatitis (Substance)

- Personal Sensitivity: Any One.

→ Substance: Irritants either physical or chemical

- EX: Acids, alkalis

Allergic contact dermatitis (Personal Sensitivity)

- Personal Sensitivity: IV hypersensitivity reaction [CMI]

- Substance: incomplete antigens i.e. haptens

EX:

- Chemicals e.g. Dyes, nickel, cement, detergents etc.
- Cosmetics: Lipsticks, nail polish, hair dyes, cycliners, depilators.
- Locally applied drugs e.g. neomycin and sulfonamides.
- Textiles: Nylon, wool, rubber, etc.

Endogenous eczema

- 1 - Atopic dermatitis
- 2 - Discoid eczema
- 3 - Pityriasis alba
- 4 - Stasis dermatitis.
- 5 - Seborrhoeic dermatitis.

Endogenous eczema

Atopic Dermatitis

Genetic factors: Immune dysregulation + Family history of allergy.

A. Infantile phase:

- Acute stage.
- Site: face
- Ill-defined erythematous patches with papulo-vesicular eruption → Exudation of serum → crusts

B. Childhood phase:

- Subacute stage
- Site: flexors.
- Erythema and scaling

C. Adulthood phase:

- Chronic stage Neurodermatitis
- Site: Back + Outer Arm + Shin of Tibia.
- Dryness and lichenification



Others

Seborrheic dermatitis

(Malassezia) (pic.c)

Types

Infantile: diaper, scalp → Cradle. Cap (up to 10 Months)

Adult: hairy areas + flexures.

Greasy yellowish scales on erythematous area (diffuse or localized)

Varicose Eczema (Stasis) Dermatitis (pic.b)

lower limbs dt (increased venous pressure)

(Discoid) Eczema (pic.A)

- bilateral -symmetrical (extensor aspects)

Inner:

erythematous scaly oozing area

Outer:

blue macules dt hemosiderin → D.2 deposition of RBCs.

Circular well

Demarcated patch.

Site

Shape

Complications

Infection

Ulceration

Immuno-dysfunction → Hyper Sensitivity

- Systemic Steroid
- Topical Steroid (subacute → Cream & chronic → ointment)

Eczema = Itching

- Eczema: emollients
- Itching: Antihistaminic
- (oozing): permanganate solution - lead subacetate solution

2ry Bact. Infection

- Systemic Ab
- Topical Ab

TREATMENT

acute stage

Dyshidrotic Eczema Pompholyx (pic.D)

on the sides of the fingers and toes and soles

face

rounded, oval hypopigmented patches with fine scales whitish

tinea pedis

MCQ

التهاب الجلد الميضي
في القدمين
H1V

يحتاج hormonal activity تاريخ
تضخم و حاد الحوصيات (الاشد حبات)
صبيانية تضخم غير كبد ال
يتضخم عند الاطفال عند سن 10 سنوات
البالغ
يظهر كدم في الجلد
scales white
Pityriasis Alba (pic.E)



URTICARIA AND ANGIOEDEMA



VASCULAR REACTION → VD → EDEMA

Site of edema:

- ^{upper} Dermis → Urticaria (wheals → dermal swelling)
- Sub.Cut + Lower Dermis → Angioedema

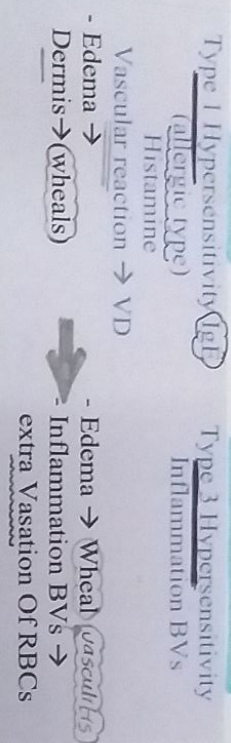
Types:

- Urticaria → With or Without Angioedema
- Angioedema → with or Without Urticaria



Urticaria

Pathogenesis



Urticarial vasculitis

Angioedema

(Histamine)
Vascular reaction → VD

- Edema:
- Dermis → Urticaria (wheals)
- Sub. Cut → Angioedema

Complications

- Systemic:**
 - Hypotension Shock → emergency
- Local:**
 - Laryngeal mucosa → emergency
 - face: Lips - Lids
 - Hands - Genitalia

Papular Urticaria

Type 4 hypersensitivity to (insect bites)

Extensor surfaces of the limbs and trunk

Prurigo of Hebra (scver)

- Insect bite reaction in atopic patients.
- Environmental factors (poor hygiene)

itchy erythematous papules → healed with hyperpigmentation

Sites

Any Where

Any Where

Clinical picture

- Wheals: Edema + Itching
- Less than 24 Hours
- With or without Angioedema

Wheals: Edema + Pain
(More than 24 Hours)

physical urticaria:

- Idiopathic
- Physical: mechanical - temperature changes - Solar - Aquagenic - Cholinergic
- Autoimmune process - infection - Drug

Duration

Acute: less 6 weeks & Chronic: More 6 weeks

TREATMENT

① Treat the Cause

- Urticarial Vasculitis or Acute
- Short course Systemic Steroid if acute

Systemic VD or Adrenaline

Adrenaline: 1/1000 subcutaneously

Antihistaminic (H1 blockers)

- 1st generation Sedating
- 2nd and 3rd G: Non-sedating

persists for several days
Recurrent

ERUPTION MOSTLY DUE TO INFECTION

ERYTHEMA MULTIFORME

Hypersensitivity Reaction

Etiology

- ✓ Infection: viral e.g., herpes simplex (MC) *← m.c.g.*
most common cause
- ✓ Idiopathic.
- ✓ Other: drugs, vaccination, malignant disease

Clinical

Symmetrical Dis: UL & LL *upper limb, lower limb*
 Target or Iris Lesion + Multiform? → erythematous macules, papules vesicles and sometimes bullae

Healing: 1-2 Weeks

Cause: Antiviral (If Recurrent: Long Term Acyclovir) *→ 6 times / year 10mg/Kg up to 6m*

Treatment

- Hypersensitivity: Sys + Topical Steroid
- Itching: Antihistaminic



ERYTHEMA NODOSUM (Pain)

Hypersensitivity Reaction

- ✓ Infection: Streptococcal infection (MC), Tuberculosis, Sarcoidosis *→ most common infection*
- ✓ Idiopathic *← most common cause → (leg)*
- ✓ Others: Drugs-Malignant diseases.

Symmetrical Dis: shine of tibia (anterior part only)

Multiple tenders, warm

Erythema → erythematous

Nodusum → nodules

Cause: Bacterial (antibiotic) ✓

Hypersensitivity: Sys + Topical Steroid ✓

Pain & Arthritis: Bed Rest & NSAID ✓

+ Sys Manifestations

most common arthritis



Steven's Johnson Syndrome

(emergency)

Severe Sys Reaction + Shedding Skin + MM

↳ *most common cause*

- **Etiology:** Medication, infection or illness
- **Clinical**
 - Shape: maculopapular → bullous → Shedding (peeling)
 - Color: Dusky red
 - Site: Generalized
 - (Painful)
 - Associations: FAHM + Arthralgia + Mucous mem. Involvement
 - Complication: Dehydration → organ failure - Infection → Death
 - Healing: without Scar
- **Investigation:** NO
- **TTT:**
 - Shedding → Stop the Drug - ICU (Burn unit) - treat the reaction
 - TTT of complication



Fixed Drug Eruption

↳ some drug cause some eruption → some site

- **Etiology:** sulfonamides and analgesics.
- **Clinical**
 - Shape: maculopapular → maybe bullous
 - Color: violaceous
 - Site: Skin + penis, lips and Mouth
 - (Painful)
 - Associations: no
 - Complication: no
 - Healing: Permanent blue discoloration ←
- **Investigation:** NO
- **TTT:**
 - Stop the drug.
 - Emollient and topical steroids



8

DISEASES OF SWEAT AND SEBACEOUS GLANDS

CONTENTS LIST

Gland secretions

Miliaria

Acne Vulgaris



SCAN TO WATCH



CHAPTER 8

DISEASES OF SWEAT AND SEBACEOUS GLANDS

• Small ductless secretory part as G, Ls
 In all body Exp: Labia minora, glans
 penis, nipple, nail bed

Eccrine sweat glands

Apocrine sweat glands

Sebaceous Glands

Holocrine Glands

Site

In all body Exp: Labia minora, glans
 penis, nipple, nail bed

During Puberty axilla - anal -
 breast - eye - external auditory

In all body Exp: Palm and Soles

Secretion

Clear Fluid

Oily Secretion

Sebum during Puberty

diseases

Miliaria

hidradenitis suppurativa

Acne

was result of obstruction of duct
 0.2-0.7 Temp



Miliaria crystalline

Miliaria rubra

Miliaria profunda

Miliaria pustulosa

Level Of occlusion of duct

At the skin surface

Intraepidermal part

Dermo-epidermal junction

Any one + 2 or Bacterial Inf

Clinical Pic

- Clear Vesicles
 - No erythema

- Papules and vesicles
 - Red halo

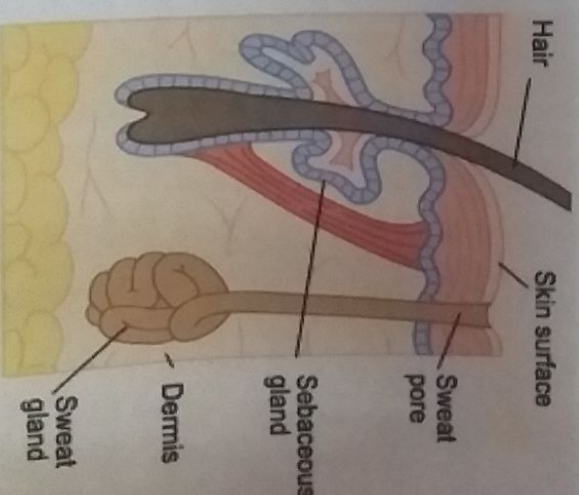
Large Papules and vesicles

Multiple Pustules

Treatment

- Cold water compresses
 - Proper ventilation

Previous + Topical AB



→ During puberty:

1. ↑ androgen secretion → activation of sebaceous gland
 → ↑ sebaceous secretion → exposure of sebaceous gland to environment → oxidation of sebaceous → open comedones (blackheads).
 2. ↑ sebaceous secretion → ↑ duct cornification → hypercornification closure of the duct → closed comedones (white heads).

SEBACEOUS GLANDS

ACNE VULGARIS

SEBACEOUS GLANDS
 (pilosebaceous units)
 puberty → Hormones → Open → Acne
 Children → Small
 Puberty (Hormones) → Hyperactivity → Large

- Org: Propionibacterium acnes (commensal)
- Clinical
 - Shape/pleomorphic →
 - ✓ Comedones: Black heads (Open) (Pic A) + White heads (Closed)
 - ✓ papules
 - ✓ pustules
 - ✓ nodulocystic (Pic B)
- Site: face, chest and back
- Itchy / painful / burn
- Asso (PDF - Sys) x
- Complication: scar
- Healing: Scar
- Investigation:
- ITT →

Pathogenesis	Hormone (Androgen)
Treatment	Antandrogens (females)
Indication	(Female)
Side Effect	Severe nodulo-cystic acne
Contraindication	Skin: Dryness ✓ MM: Dryness ✓ Hair: Loss ✓ Ms: Pain ✓ Liver Enzyme: ↑ ✓ S. lipid: ↑ ✓

Pregnancy



Post acne scars treated by:
 dermabrasion, chemical peeling or laser resurfacing

Cornification
 1. Retinoids
 2. sulfur ppt
 3. salicylic acid
 4. azelaic acid
 Propionibacterium acnes
 1. Tetracycline
 2. Doxycycline
 3. Erythromycin
 4. Clindamycin
 Retinoids + antibiotics
 Mild to moderate " 1 course

9

Erythematous squamous eruptions

CONTENTS LIST

Psoriasis (الصدفية)

Variants of psoriasis (المتغيرات)

Pityriasis rosea (الطفح الوردي)

Lichen planus

Types of lichen planus



SCAN TO WATCH

PART 1



SCAN TO WATCH

PART 2



- ✓ Psoriasis
- lichen planus
- pityriasis rosea

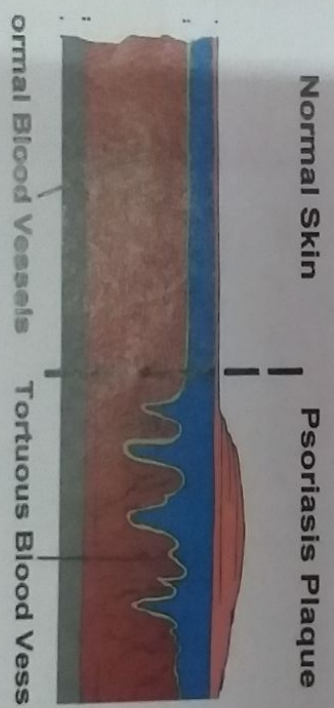
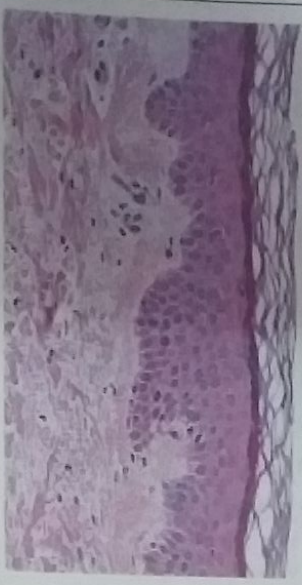
CHAPTER 9

ERYTHEMATOUSQUAMOUS ERUPTIONS

(التهاب الحويصلات) **PSORIASIS**

unknown initiator!! → Inflammatory Process
 rapid proliferation → affect differentiation

التهاب الحويصلات هو التهاب مزمن في الجلد
 يتميز بآفات غير حادة في الجلد، في الجلد
 (epidermis) تتميز بـ: جودة الجلد
 more superficial (على سطح الجلد) →
 dense scales (قشور كثيفة) (التهاب الحويصلات)



- * ortho keratosis :-
normal horny cells
- * para keratosis :-
abnormalities in horny cells (presence of nuclei)
- * acanthosis :-
epidermal thickening
- * papulo-dermatosis :-
dermal papule



PSORIASIS → (3% of population)

A chronic inflammatory, non-infectious skin disease characterized by well-defined erythematous papules and plaques covered by silvery laminated scales

(paronychia)

Psoriasis: disease of adults with 2 exceptions: napkin & guttate ps

scheme:-

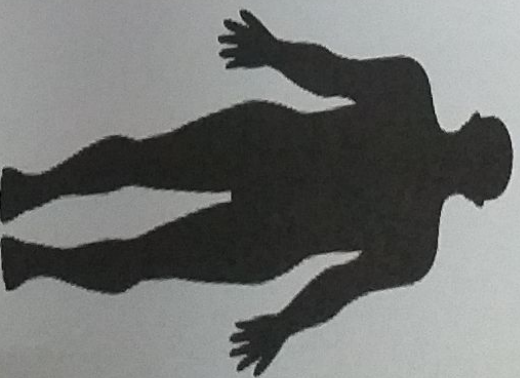
Variants:

- Name
- Site
- Shape
- Specific → severity

Etiology: unknown

Clinical:

- Shape: multiple (erythematous papules) and (plaques) - silvery laminated scales
- Site: extensors - (trunk)
- Mild itch
- Asso (PDE - Sys)
- Complication
- Healing: usually leave hypopigmented
- Investigation: Grattage test - Biopsy.
- TTT:



Predisposing Factors:

- Trauma (Koebner phenomenon) → Development of psoriatic lesions commonly occurs at the site of trauma
- Infection: post streptococcal → infants after tonsillitis → guttate psoriasis.
- Pregnancy
 - Improve generalized psoriasis
 - provokes generalized pustular psoriasis.
- Climate: sunlight → improve psoriasis
- Metabolic Factors: (Hypocalcemia)
- Drugs: Antimalarials, beta-blockers and lithium
- Stress: trigger factor.

Investigation:

- Grattage test: Scraping lesions with glass slide remove the scales → pin-point bleeding → [Auspitz sign]
- Biopsy

Topical and systemic steroids → erythrodermic or pustular psoriasis.

MOST SEVERE FORMS

Erythrodermic psoriasis

Caused by severe infection - hypocalcemia - Systemic Steroid

Shape More 90% of skin scales and Erythema

Site Extensive → all the body



Arthropathic psoriasis

↓ less disability

Pustular psoriasis

Systemic Steroid
0-2 neutrophilic infiltration
(Erythema and Pustules)

Types:

- Generalized
- Localized to palm and soles



With or without Cutaneous Psoriasis

May be:

- Distal interphalangeal joint
- Oligoarticular or polyarticular



TREATMENT

Selection of the treatment according to:

- Extent of the disease
- Patient's perception of the severity
- Potential side effects of the specific treatment

INITIATOR! → INFLAMMATORY → PROLIFERATION → UNDIFFERENTIATION → SCALES

TOPICAL TREATMENT

Emollients

Keratolytic → the scales e.g. **salicylic acid** (3 - 10%)

Tar preparation e.g. crude coal tar 2 - 5%.

Action: anti-proliferative

S/E: Unpleasant odor and staining of cloths.

Topical steroid

Intralesional steroid > small resistant lesions - nails.

Vitamin D3 analogues

Action: inhibit cell proliferation and stimulate keratinocyte differentiation

S/E: Irritation and hypercalcemia

in pregnancy, lactation and renal insufficiency

Topical retinoids as Tazarotene gel.

Anthrallin [Dithranol]:

Action: antimitotic effect.

S/E: irritant and stains skin

Potent steroids should be avoided on face, flexures and in young patients

PHOTOTHERAPY

Alone: **UVB** (narrow band or broad band).

UVB is safe in children, pregnancy and lactation.

(Narrow band UVB) is the mainstay of treatment for moderate to severe psoriasis

Combined: Photochemotherapy

UVA (psoralen + UVA): effective but with long-term risk of skin cancer.

SYSTEMIC THERAPY (MAC)

Indications:

- Generalized pustular, erythrodermic or arthropathic psoriasis.
- Resistant to topical therapy.
- Psychological distress

(M) Methotrexate: Folic acid antagonist.

Indicated: listed b4

Dose: 0.2 - 0.4 mg/Kg weekly

S/E: hepatotoxicity and BM suppression.

pregnancy and liver diseases

(A) Acitretin (etretinate): sys vit A derivative.

Indicated: listed b4

Dose: 0.5 - 1 mg/Kg/day.

S/E: teratogenicity and increase lipid level.

children and pregnancy.

(C) Cyclosporin A: immunosuppressive.

Indicated: listed b4.

dose: 2.5 - 5 mg/Kg/day.

S/E: hypertension and nephrotoxicity

-Others: Biological therapy: as tumor necrosis factor inhibitors.

= contraindicated

PITYRIASIS ROSEA

(Oligosymptomatic)

• Etiology:

- Unknown
- Viral (HHV6, HHV7)

• Clinical:

- Shape:
 - Herald patch → Single (erythematous oval patch) with fine (collarette of scales)
 - 2nd Rash → After 2 weeks → Small generalized symmetrical erythematous maculopapular rash.

▪ Site:

- Trunk - Neck - Arms

→ Christmas tree pattern distribution

- Itchy in some cases.

- Healing: after 6 - 8 weeks

▪ DD:

- Tinea circinata (herald patch).
- Guttae psoriasis
- Secondary syphilitic rash.
- Pityriasis drug eruption.

• Investigation: No

• TTT:

- Self-limited
- Reassurance
- Avoidance of irritants
- Soothing lotion + topical steroid
- acyclovir ← (viral causation)



Variants:

- Abortive type: Herald patch appears only + No 2nd rash.
- Inverted type: Face and limbs are affected sparing the trunk

(least papules without scales) LICHEN PLANUS (systemic) UNKNOWN INITIATOR → IMMUNO-DYSFUNCTION → INFLAMMATION → ITCHY PAPULES

• Etiology:

- unknown
- viral (HCV)
- Autoimmune
- Malignancy.

• Clinical

- Shape: multiple flat-topped polygonal papule
- Color: violaceous with whitish striations called Wickham's striae
- Site: wrists, forearms, legs and genitalia
- Severely Itchy
- Predisposing factors: trauma (Koebner phenomenon)
- Complication -
- Healing: hyperpigmentation

• Investigation: Biopsy

• TTT: Unknown

- Viral → Treat Of virus. ^{HCV}
- Sun Block → ^{topical} Treat Of virus.
- Immuno-dysfunction → Steroid and anti-malarial.
- Inflammation → Topical and Intra-lesional And Systemic steroid
- Itchy papules → Anti-histamines.



OTHER TYPES OF LICHEN PLANUS

(HAIR – MUCCOUS MEMBRANE – SKIN – NAILS)

Lichen planopilaris

Site
(Hair follicles on the scalp)

Cause **cicatricial alopecia**

Graham Little syndrome:

- triad
1. **Scalp** cicatricial alopecia
 2. **Non scarring** alopecia of axillae and/or groin
 3. keratotic lichenoid follicular papule)



Lichen planus of the mucous membrane

- Buccal mucosa + lips
- Glans penis
- Vulvovaginal region
- Anus

→ (Wickham's striae)

Ulcers may turn malignant in 1-2% (squamous cell carcinoma)



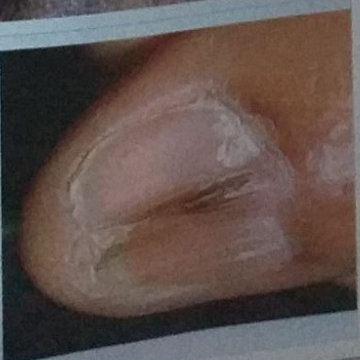
Skin

- (hypertrophic) lichen planus, shin of the tibia
- Hypertrophic lichen planus, shin of the tibia
- Actinic lichen face (sun-exposed areas)
- Acute (eruptive) lichen planus
- Atrophic lichen planus.
- Annular lichen planus.
- Linear lichen planus.
- Bullous lichen planus.

Lichen planus of the nails

- pterygium (pic)
- Longitudinal ridges
- pitting
- Shedding of the nail

Lichenoid drug eruption: Gold, antimalarials, para-amino salicylic acid, and streptomycin



10

DISEASES OF THE HAIR

CONTENTS LIST

Hair Follicle

Types of alopecia

Alopecia areata

Other variant of non cicatricial alopecia



SCAN TO WATCH



Hair is present all over the body except the palms, soles, dorsal aspect of terminal phalanges of digits, glans penis, labia minora, lips, eye lids, areola and mucocutaneous junctions

CHAPTER 10 DISEASES OF THE HAIR

Hair Follicle

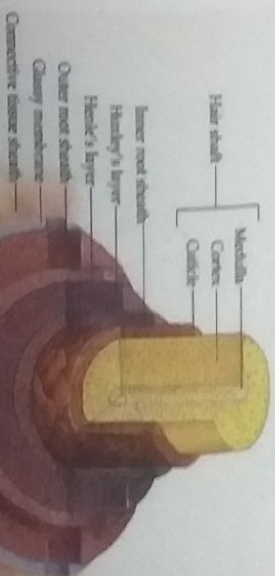
- Hair shaft → centre of hair follicle.
→ in its centre there is [medulla] covered by
- Outer root sheath
- Inner root sheath

Types of Hair Follicles:

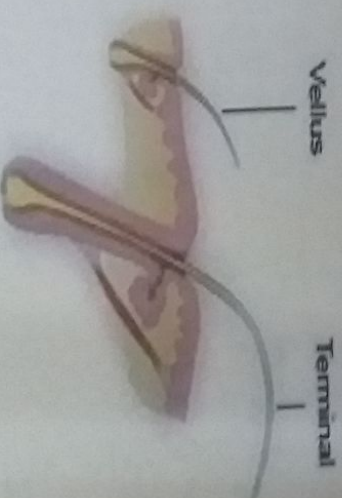
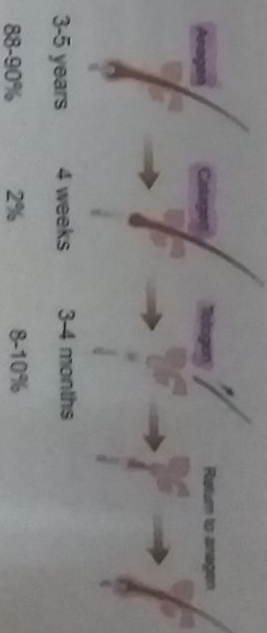
- 1- Lanugo (infants) → soft, thin hair without medulla.
- 2- Vellus (Youngers) → soft, thin hair without medulla.
→ (axilla + pubic area)
- 3- Terminal (Adults) → with medulla.

Hair Cycles: (stages of hair growth)

- ACT → Anagen 3y → Catagen 3w → Telogen 3m



Stages of Hair Growth



ALOPECIA LOSS OF HAIR

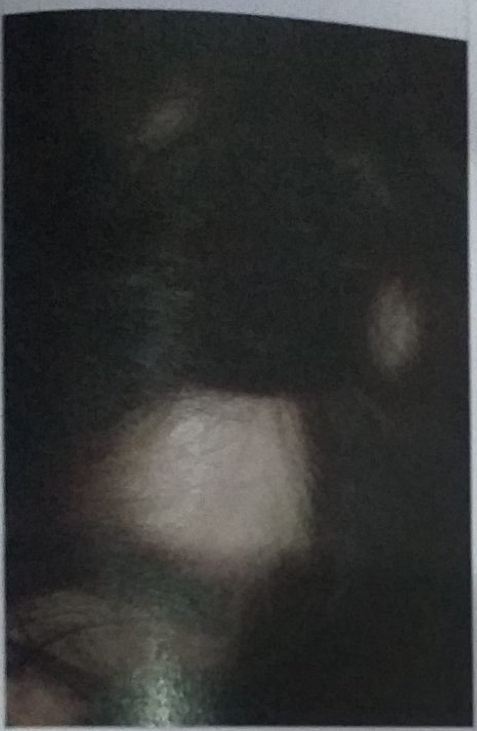
Hair Follicles Healing Hair growth	Non-cicatricial alopecia		Cicatricial alopecia	
	Healthy (Not Damaged)		Damaged	
	Non scar		Scarring	
	Regrowth		No growth	

Localized non-cicatricial alopecia:

- Alopecia areata.
- 1- Male baldness. (*androgenic alopecia*)
- 2- Trichotillomania.
- 3- Tinea capitis

Diffuse non-cicatricial alopecia:

- A- Stress conditions: telogen effluvium – Psychogenic
- B- Endocrinal: hypo- and hyper-thyroidism, hypopituitarism.
- C- Anemia and malnutrition.
- D- Debilitating diseases: e.g. malignancies.



Trauma: physical or chemical.

Tumor: basal cell carcinoma (cyst tumor)

Infections:

- 1- Bacterial: staphylococcal infections, lupus vulgaris,
- 2- viral infections: herpes zoster
- 3- Fungal: Kerion and Favus

Autoimmune as DLE and scleroderma – Lichen P.

Idiopathic



ALOPECIA AREATA

AUTOIMMUNE → HAIR FOLLICLES → SUDDEN LOSS HAIR + NORMAL SKIN

سقوط مفاجئ للشعر مع بقاء الجلد طبيعياً

Def: sudden loss of hair in a circumscribed area (or areas)

Etiology: Genetic- Immunological

Clinical: ^{most acceptable}

- Shape: Sudden Loss of hair + exclamation mark hairs thin proximally and thick distally → indicates progression of the disease
- Color: normal skin with no crusts or scales
- Site: Any hairy area
- Types
 1. Patchy: most common
 2. Alopecia (Totalis): affect the hair of all scalp
 3. Alopecia (Universalis): affect the hair of all body

- Asymptomatic

- Associated with: ocular – dental origin – autoimmune diseases.

- Healing: No scars and regrowth thin, fine and colorless hairs (نمو شعر جديد رقيق)

Poor prognosis: Prepubertal - posterior scalp - atopy (مخاطر عالية)

Diagnosis: clinically

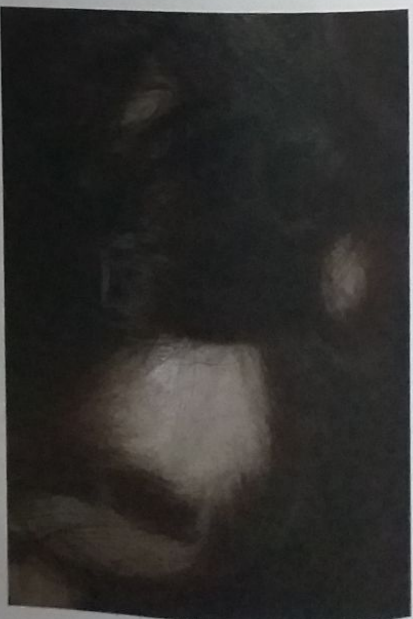
TTT:

Immunological: topical – intralesional steroid – Systemic Steroids

Irritation for hair follicles: (Iodine, Jodine)

Both: DPCP – Topical PUVA

Others: camouflage – treatment septic focus



OTHER VARIANT OF NON CICATRIAL ALOPECIA (أشكال أخرى من الصلع غير الندبي)

Androgenetic Alopecia (الصلع الوراثي)

* Androgen dependent

convert hair follicles from terminal to vellus

Age: start after second decade post-puberty

Sex: male and female

Site:

- Sensitive to Androgen: frontal, parietal & crown

- Resistant: occipital and temporal

Trichotillomania (اضطراب قلع الشعر)

compulsive habit neurotic individuals

Telogen Effluvium (الصلع المتقطع)

(Stress) conditions

labor, operations, shock, fever

Hair follicles: prematurely enter the telogen phase

Duration: 3 - 4 months after exposure to stress.

Psychiatric consultation

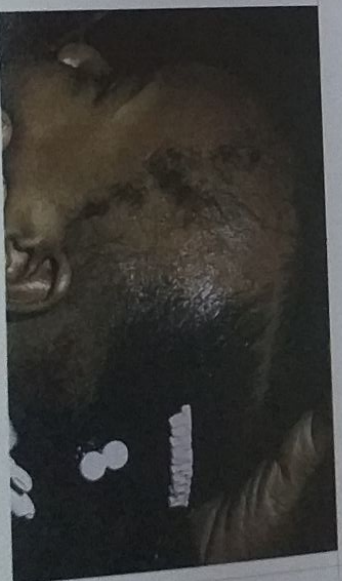
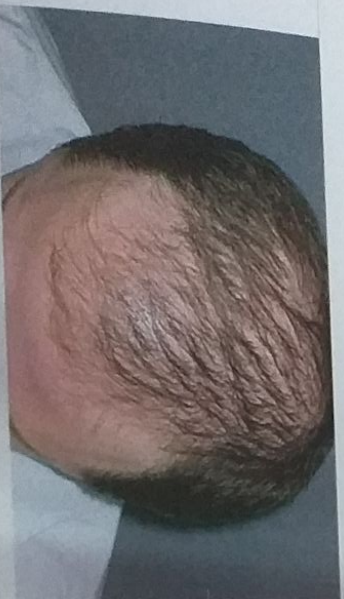
good prognosis and hair re-growth occurs within 3-4 months.

Local minoxidil 2-5%

Males: Finasteride 1mg/day

(selective type II 5-alpha reductase inhibitor).

Female: Systemic anti-androgens



11

Other skin disorders

CONTENTS LIST

PIGMENTARY DISORDERS OF THE SKIN. (vitiligo – melasma).
CONNECTIVE TISSUE DISEASES. (discoid lupus).
BULLOUS DISORDERS. (pemphigus vulgaris – bullous pemphigoid).



SCAN TO WATCH

PIGMENTARY DISORDERS



SCAN TO WATCH

Discoid lupus & bullous diseases



PIGMENTARY DISORDERS OF THE SKIN

Color of the skin

- Red: oxygenated Hg
- Blue: Deoxygenated Hg
- Yellow: carotene from diet
- Brown: Melanocyte (skin)

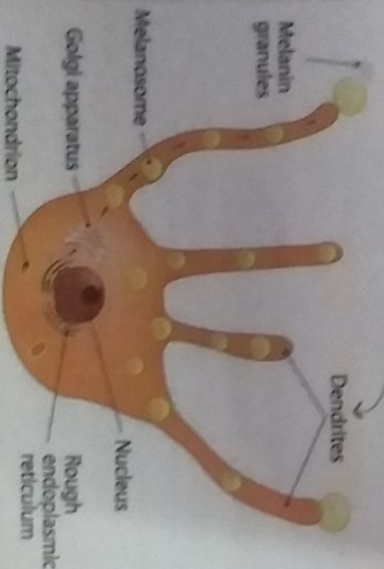
Melanocytes

Site:

Epidermis, hair follicles, eyes, leptomeninges and inner ear.

Function:

- Melanogenesis: biosynthesis of melanin in melanosomes where the tyrosine converted to melanin by (tyrosinase) enzyme
- Distribution and transfer of (melanin) to keratinocytes.



HYPOPIGMENTATION

1- Localized

- 1- Melanocyte defect
- Vitiligo (autoimmune)

2- Generalized

- 1- Melanocyte defect
- Vitiligo (autoimmune)

HYPERPIGMENTATION

1- Localized

- Post-inflammatory: (melanin)
- Iichen planus.
- Melasma (melanin)
- Naevi (melanocytes)
- Drugs:**
- Estrogen.
- Oral contraceptives.
- Tetracyclins
- minocycline.
- Chloroquine

2- Generalized

- Addison's Disease:**
- Generalized.
- Buccal mucosa.
- palmar creases.
- flexures, scars.
- Cushing syndrome**
- the trunk, upper arms.

1 Melanin defect:

- Pityriasis versicolor
- Post inflammatory hypopigmentation
- Tubercloid leprosy

2 Melanin defect:

- Albinism (genetic)
- hypopigmentation (endocrine)

VITILIGO

Etiology:

1. **Positive family history.**
2. Auto-immune: associated with $\rightarrow$ Steroid $\rightarrow$ autoimmune thyroid disease, alopecia areata, Pernicious Anemia $\rightarrow$ *Exacerbated by stress, UVB*
3. Neurogenic and Psychogenic factors.
4. Melanocyte self-destruction theory.

> Def: idiopathic chronic disorder $\rightarrow$ Destruction of melanocytes $\rightarrow$ pigmentary loss

> Clinical:

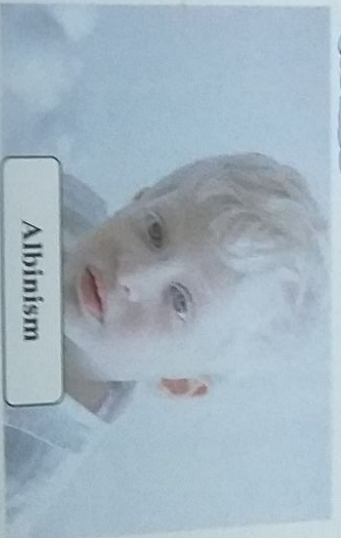
- Shape: Milky or chalk white (*depigmented*) macules, patches associated with (Koebner's phenomenon)
- Site: any Area
- Asymptomatic, *
- Complication: NO *
- DD:
 - Pityriasis alba.
 - Tuberculoid leprosy.
 - Post-inflammatory hypopigmentation.
 - Pityriasis versicolor.
 - Albinism.
 - Nevus anemicus (*defect in blood vessels*)

> Diagnosis: clinically

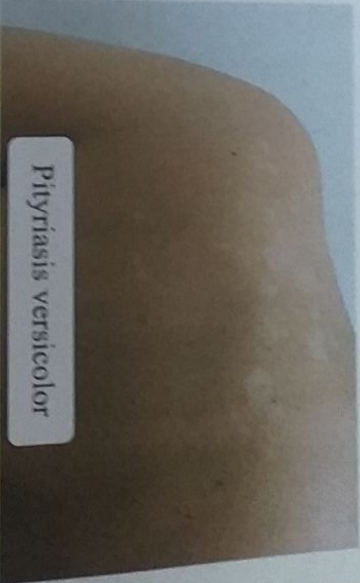
> TTT:

Inflammatory Process: Steroid + Phototherapy

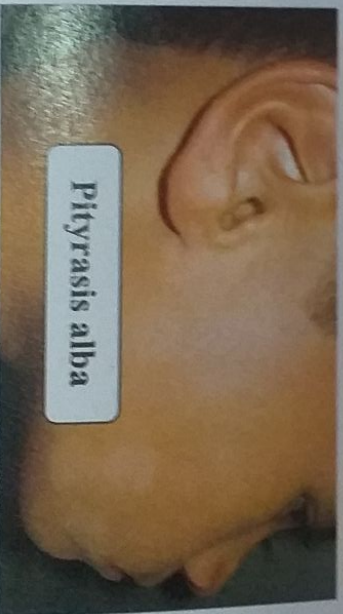
1. Steroid topical or systemic $\rightarrow$ *resistant $\rightarrow$ progression*
2. phototherapy: PUVA - NBUVB
3. Non-Response: minigraft surgical



Albinism



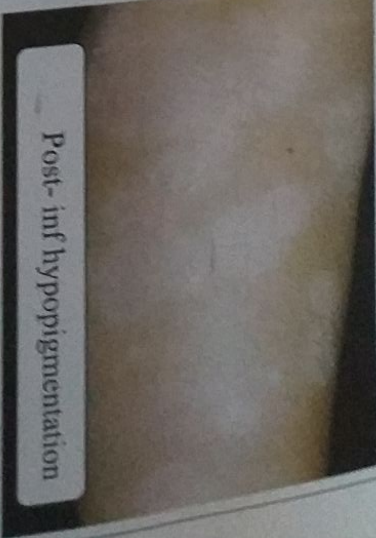
Pityriasis versicolor



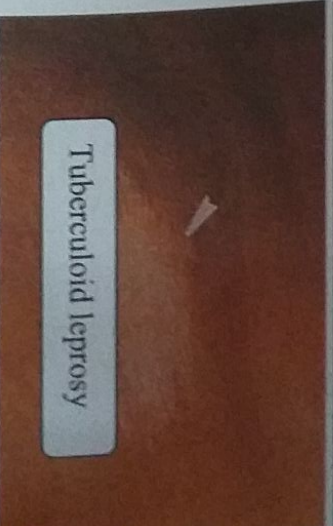
Pityriasis alba



Nevus anemicus:



Post- inf hypopigmentation



Tuberculoid leprosy

> Def: acquired hyperpigmentation disorder

> Etiology: *(melanin defect)*

Sun + ↑ Hormones (pregnancy -OCP - Ovarian Cancer)

> Clinical

- Shape: Well-demarcated hyperpigmented (macules) & Patches)
- Site: cheeks, forehead, nose upper lip and chin
- Asymptomatic
- Associated with → ↑ hormones
- Complication: NO
- DD:

• Actinic lichen planus

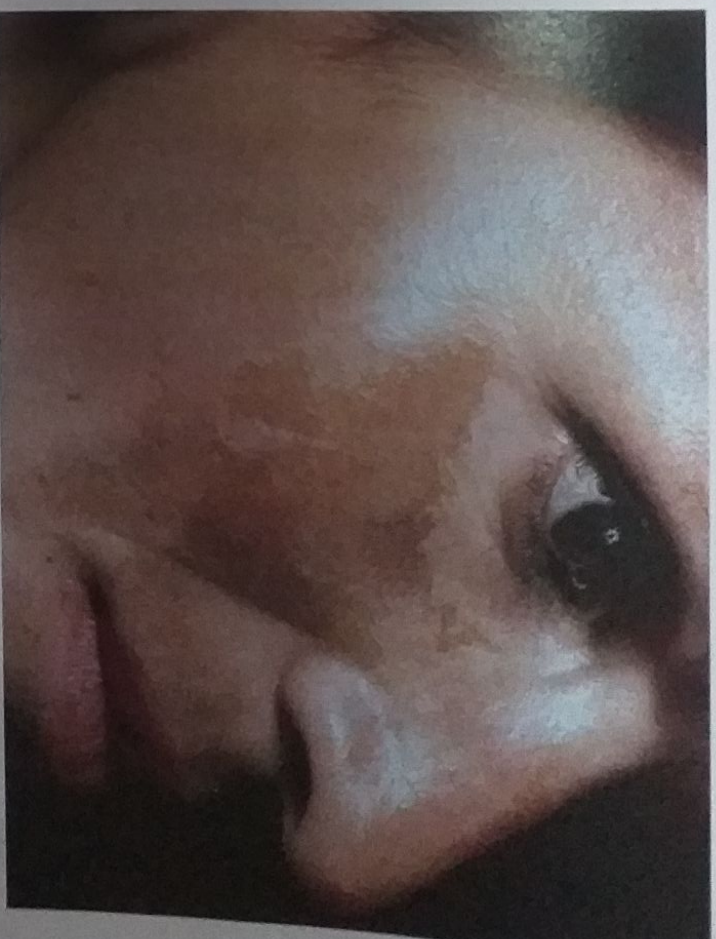
• Post inflammatory hyperpigmentation

> Diagnosis: clinically

> TTT:

(Sun + ↑ Hormones → activate Melanocytes → Pigmentation)

1. Sun Block
2. Stop OCP
3. *Hydroquinone, azelaic acid. → ↓ activity ↓ pigmentation*
4. Chemical peeling using trichloric acid



autoimmune $\rightarrow$ Genetic factors
 $\rightarrow$ Environmental factors

CONNECTIVE TISSUE DISEASES

Acute Cutaneous LE

Mainly systemic disorder + skin

Subacute Cutaneous LE

In-between

Subacute Cutaneous LE

Mainly cutaneous disorders
 Mainly female
 3rd decades

5% biopsy positive 5% clinical
 acute CI

Discoid lupus = chronic cutaneous lupus E.

Definition:

Clinical:

Shape:

(Coin-shaped plaques) with adherent Scales and dilated hair follicles with telangiectasia

Edge is (hyperpigmented) - erythematous central

Site: Localized (Head) Disseminated

- Asymptomatic
- Associated with SLE in 5%
- Healing: Thin Atrophic scar $\rightarrow$ cicatricial alopecia.

DD:

- Lupus Vulgaris
- Psoriasis

Diagnosis: biopsy



TT:-
 - sun block (topical)
 - " " systemic
 - Steroid (intra-lesional)
 - anti-inflammatory
 - e.g. anti-malarial

BULLOUS DISORDERS

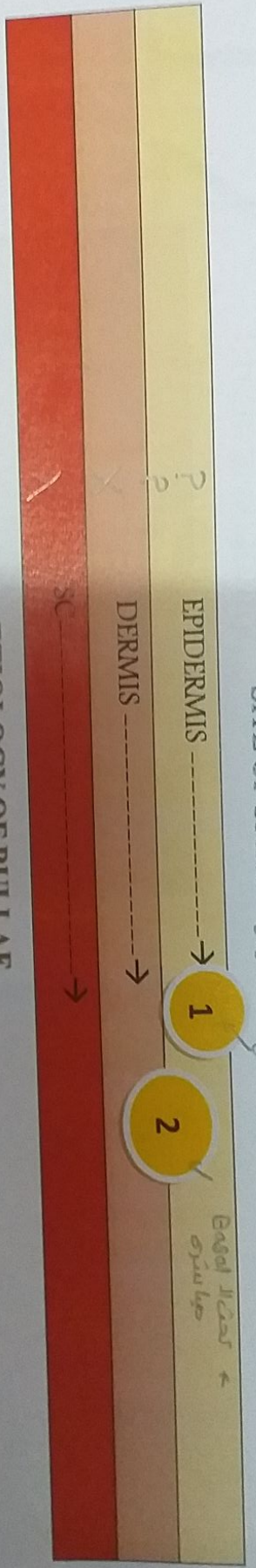
lesions filled with fluid form in the superficial layers of skin

1ry lesion

Vesicles or bullae

2ry lesion

Erosions + crusting



ETIOLOGY OF BULLAE

1. Congenital	2. Infections	3. Physical injury	4. Drugs	5. Autoimmune	6. Other
→ Epidermolysis bullosa → Fungal: Tinea pedis	→ Bacterial: Impetigo → Viral: Chicken pox, Herpes → Fungal: Tinea pedis	Cold Heat (sunburn) Friction	→ Fixed-drug eruptions	Pemphigus Bullous pemphigoid	→ Eczema Erythema multiforme Vasculitis

above basal cell layer.

1. Epidermis → flaccid bullae → easily ruptured → lesions mainly erosions ((Pemphigus))

2. Dermo-epidermal junction → tense bullae → not easily ruptured → lesions bullae and erosions ((Bullous pemphigoid))

below basal cell layer.

DIFFERENCE BETWEEN PEMPHIGUS VULGARIS AND BULLOUS PEMPHIGOID

	Pemphigus vulgaris	Bullous Pemphigoid
Etiology	Autoimmune	Autoimmune
Site of Split	Above Basal (Epidermis)	below the basal layer (DEJ)
Skin	Flaccid blisters (easy Rupture) → erosions	Urticarial (itching) → tense bullae → erosions
Mucous Mem	100%	30%
Specific	+ve Nikolsky sign → (extensive) Tense Bullae	-ve Nikolsky sign
Investigation	Skin (Biopsy) Split above Basal (AB) From lesion (Periphery) – blood	Skin Biopsy: Split below Basal + Eosinophils → Urticarial AB: From lesion (Periphery) – blood
TTT	Steroid (60 to 120 Mg) + other immunomodulators	Steroid (up to 60 Mg) + other immunomodulators



Nikolsky sign: skin at the edge of a blister slides off when pushed by a finger.



12

URETHRITIS

CONTENTS LIST

Urethral anatomy

Types of urethritis

GONORRHEA

CHLAMYDIA TRACHOMATIS

Vulvovaginitis. (TRICHOMONIASIS)

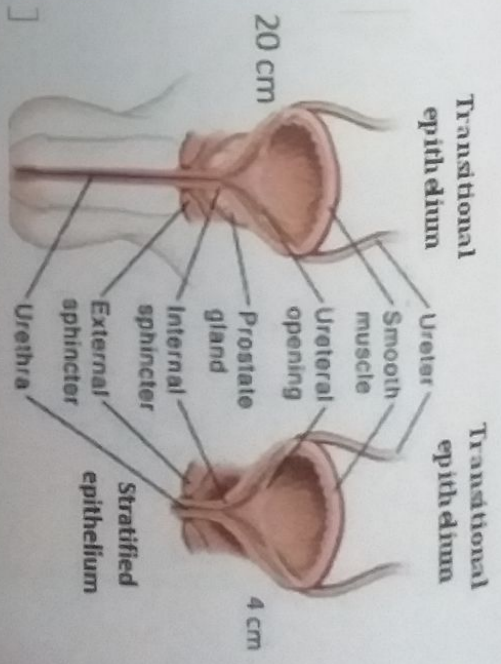


SCAN TO WATCH

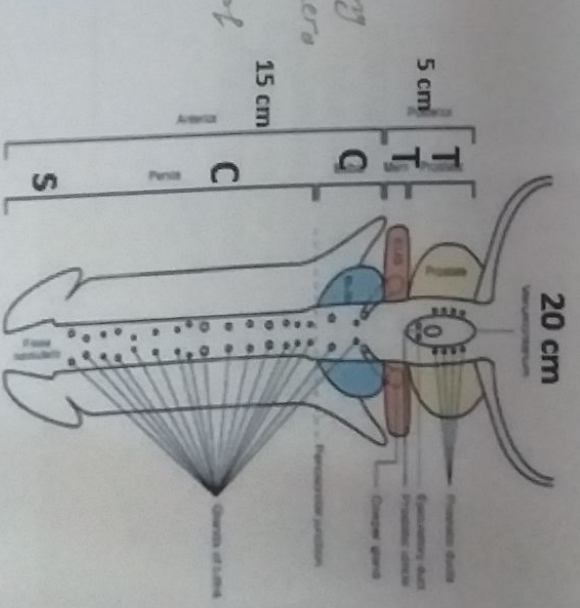


CHAPTER 12 URETHRITIS

	Anatomy of the female urethra	Anatomy of the male urethra
Length	4 cm	20 cm
Lined by	Px: transitional epithelium Ds: stratified epithelium	Two Parts
Glands	Skene's glands	Will be mentioned



	Posterior urethra	Anterior Urethra (spongy-penile)
Divided	Prostatic urethra Membranous urethra	Bulbous Urethra Distal or pendulous Urethra
Parts: From - to	F: prostatic apex T: penile bulb	F: triangular lig T: penile angle F: penile angle T: fossa navicularis
Length	5 cm	15 cm
Glands	Prostatic acini + common ejaculatory duct	Cowper's G duct (open)
Epithelium	Transitional epithelium	Columnar Epithelium In (fossa navicularis)



Urethritis

Gonococcal

Due to *Neisseria gonorrhoeae*

Scheme:

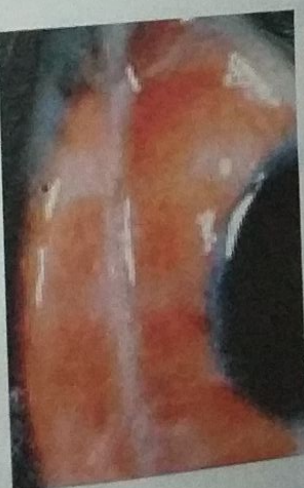
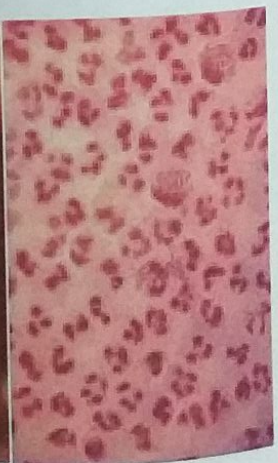
- Organism -
- Mode of infection - *sexual*
- Incubation period. - *discharge - 1-2 days*
- Clinical Picture:
 - Urethritis (anterior - posterior)
 - *MM - abnormal smears*
- Complications:
 - Glands open in urethra
 - Sys *inf.*
 - Abnormal age
- Diagnosis: Smear - culture - immune - PCR.
- Treatment according to organism

Non gonococcal

1. Sexually transmitted:
 - Chlamydia trachomatis (main cause)
 - Mycoplasma and Ureaplasma urealyticum.
 - Trichomonas vaginalis
2. Non-sexually transmitted:
 - Urinary tract infection
3. Secondary to: urethral instrumentation:
 - foreign bodies, renal stones or crystalluria



- ← بئزلا اهل حال على سقزته
معايش بيقط حيا من
سقزته ده



Complications:

Local complications:

Male:

- Balanitis or balanoposthitis, tyrositis.
- Littatus, peritumoral abscess, Cowperitis.
- Prostatitis (acute, chronic, abscess).
- Chronic prostatitis → 10 pus cells / high power field, with clumps.
- Seminal vesiculitis (painful erections and haemospermia).
- Epididymitis.
- Urethral stricture

Female:

- Skentitis
- Bartholinitis.
- Pelvic inflammatory disease (PID).
- Phlepatitis in some cases with salpingitis
- **Antiovaaginitis** not common in adult female

extension from vagina to endometrium, fallopian tubes, ovaries, Peritonium.

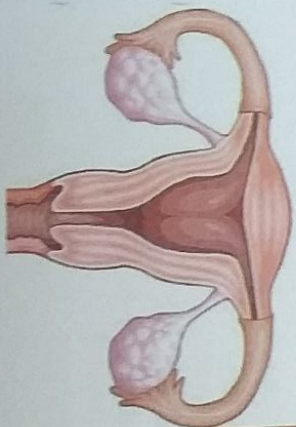
can premature female

Systemic complications: Disseminated gonorrhoea

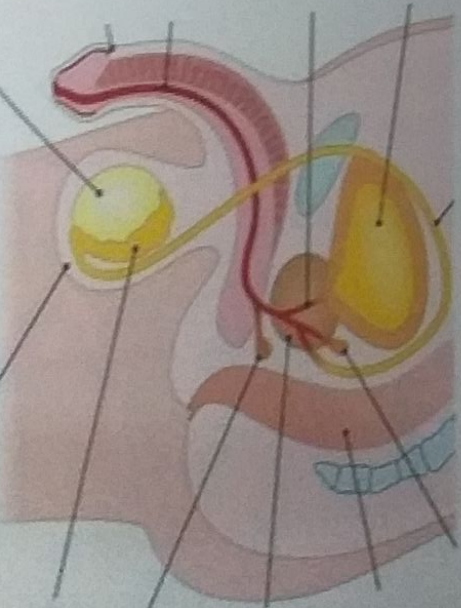
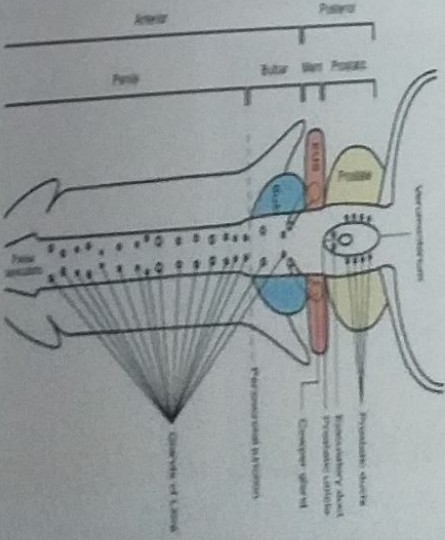
- More in females ← as it's asymptomatic.
- Fever, joint affection, and skin rash
- Blood cultures? usually positive

(Age) Gonorrhoea in children: Gonococcal vulvovaginitis

- Accidental or sex abuse.
- Immature stratified squamous epithelium.
- vaginal discharge, itching and dysuria.
- Gonococcal ophthalmia IP? → (3-5) days



Chlorea
Labia minora
Urethra
Bartholin's glands
Vagina



(التهاب المفاصل الجرثومي)

Diagnosis:

• Smears:

- Site: Discharge - Morning Drop - over/night urine (in contacts)
- Stain: Gram Stain → negative diplococci

• Culture:

- Indication: Asymptomatic patient
- Type of culture:
 - Growth medium: Chocolate agar
 - Selective medium: Thayer Martin medium

- Immunological: Direct immunofluorescence - Radioimmunoassay - Monoclonal antibodies technique

- Tests for antibiotic sensitivity: Disc Method

Treatment:

I. Systemic antibiotics:

2 1 400 250

1. Spectinomycin: 2 SD (first line of treatment).
2. Azithromycin: 1 gm orally SD.
3. Quinolones: Ofloxacin 500 SD. (400)
4. Ceftriaxone: 250 SD
5. Penicillin:
 - Penicillin interferes with the cell wall synthesis.
 - Procaine penicillin 4.8 million units IM at 2 sites.
 - Ampicillin 3 gm single dose.
 - Amoxycillin 3 gm single dose.

(علاج)

II. Abstain from alcohol and sexual intercourse.

NB: (increased resistance from Gonococci has been recently demonstrated)

Follow up

At the 3rd day of treatment there will be no more discharge, and this means

a successful treatment

@AUBdatabot

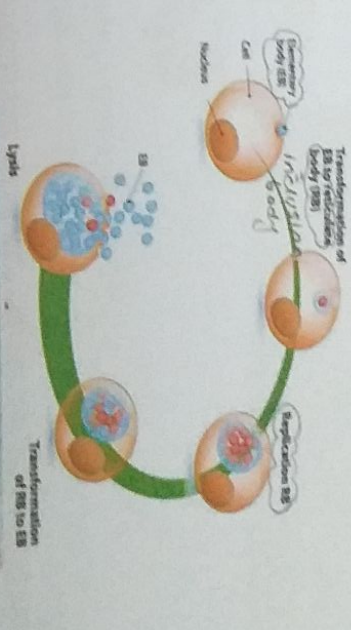
* main cause of non-gonococcal

CHLAMYDIA TRACHOMATIS

OBLIGATORY INTRACELLULAR

causative organism (culture below)

LIFE CYCLE OF THE CHLAMYDIA



- > **Causative organism:** Chlamydia trachomatis
 - No artificial media, but yolk sac and chick embryo.
 - Elementary body, (infection)
 - inclusion body (replication)

> **Incubation period:** 2 - 3 weeks (Post gonococcal urethritis).

> **Mode of infection:** Sexual

> **Clinical picture:**

Urethra

• **Anterior Urethra:** Discharge

- Type: mucopurulent
- Amount: scanty (القلي)
- Color: white to yellow (عسل)

• **Posterior Urethra:** Dysuria, Frequency and Urgency.

Other MM (Direct Contact):

- Anal: Proctitis

Complications:

Local complications: follow Anatomy *Perix 1/2, 2/2, 3/2, 4/2, 5/2, 6/2, 7/2, 8/2, 9/2, 10/2, 11/2, 12/2*

Distant and systemic complications: **Reiter's syndrome**

- **Conjunctivitis:** can't see.
- **Urethritis:** can't pee
- **Arthritis:** can't climb the tree.

(Age) Neonatal Chlamydial infection

conjunctivitis - pneumonia - otitis media.

IP 1/2, 2/2, 3/2, 4/2, 5/2, 6/2, 7/2, 8/2, 9/2, 10/2, 11/2, 12/2

Blood Culture

reaction (claydria)



Diagnosis:

(Obligatory intracellular organism → no artificial media, but yolk sac and chick embryo)

- Smears:

- Site: Discharge
- Stain: immunofluorescence (anti-Chlamydial AB)
* labell'd Ig → Ag ultrastructural
- Culture: Isolation on living cells.

- Immunological: Direct immunofluorescence.

anti-chlamydial AB

- PCR.

Treatment

TE DATA

→ contra indicated in pregnancy.

- Tetracycline 500 mg 1×4×14.
- Erythromycin 500 mg 1×4×14. (Pregnancy)
- Doxycycline 100 mg 1×2×14.
- Azithromycin 1 gm single dose. (Pregnancy)

First line for any discharge
gonorrheal & chlamydia
safe in pregnancy

MYCOPLASMA AND UREAPLASMA UREALYTICUM:

- Etiology: contains both DNA and RNA
- Clinical picture and complications: The Same
- Laboratory: SCI (mycoplasma grows on artificial media)
- Treatment: Same as B4

[Female] - **VULVOVAGINITIS**

TRICHOMONIASIS

1. Bacterial vaginosis
2. Trichomonas vaginalis
3. vaginal candidiasis

Causative organism: *Trichomonas vaginalis*

- Anaerobic protozoan.
- Motile

Incubation period: 3 days – 3 weeks.

Mode of infection:

- Sexual intercourse.
- Non-sexual transmission through fomites.

Clinical picture:

Females:

• **Vaginitis; discharge**

- Amount: profuse *gonorrhea like*
- Odor: *offensive* discharge

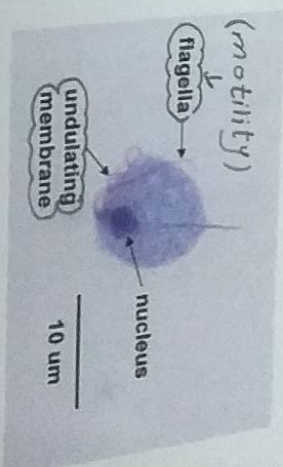
- Cervix: red spots on cervix (*strawberry cervix*).
- Urethritis.

Males:

- Asymptomatic urethritis
- A tickling sensation in the urethra

Diagnosis:

- Direct microscopic examination (*wet mount preparation*) (*saline + discharge*)
- Stained smear (less reliable): Papanicolaou Stain.
- Culture.



Treatment (anaerobic protozoan):

- Metronidazole 250 mg 1x3x7^{days}
- Tinidazole 2 gm single dose.
- Treatment of the sexual partner

13

SYPHILIS and other STDs

CONTENTS LIST

General scheme

- Acquired syphilis
- Difference between condyloma accuminata and condyloma lata
- Latent syphilis
- Tertiary stage (Gumma)
- Congenital Syphilis
- Investigations and treatment

OTHER SEXUALLY TRANSMITTED DISEASES



SCAN TO WATCH



CHAPTER 13 SYPHILIS

Organism: *Treponema pallidum*

Mode of infection:

Acquired:

- Sexual transmission: **Most common**
- Accidental: direct (doctor), indirect (toilet seats) (very rare)
- Fresh blood transfusion: if the donor is infectious.

Congenital:

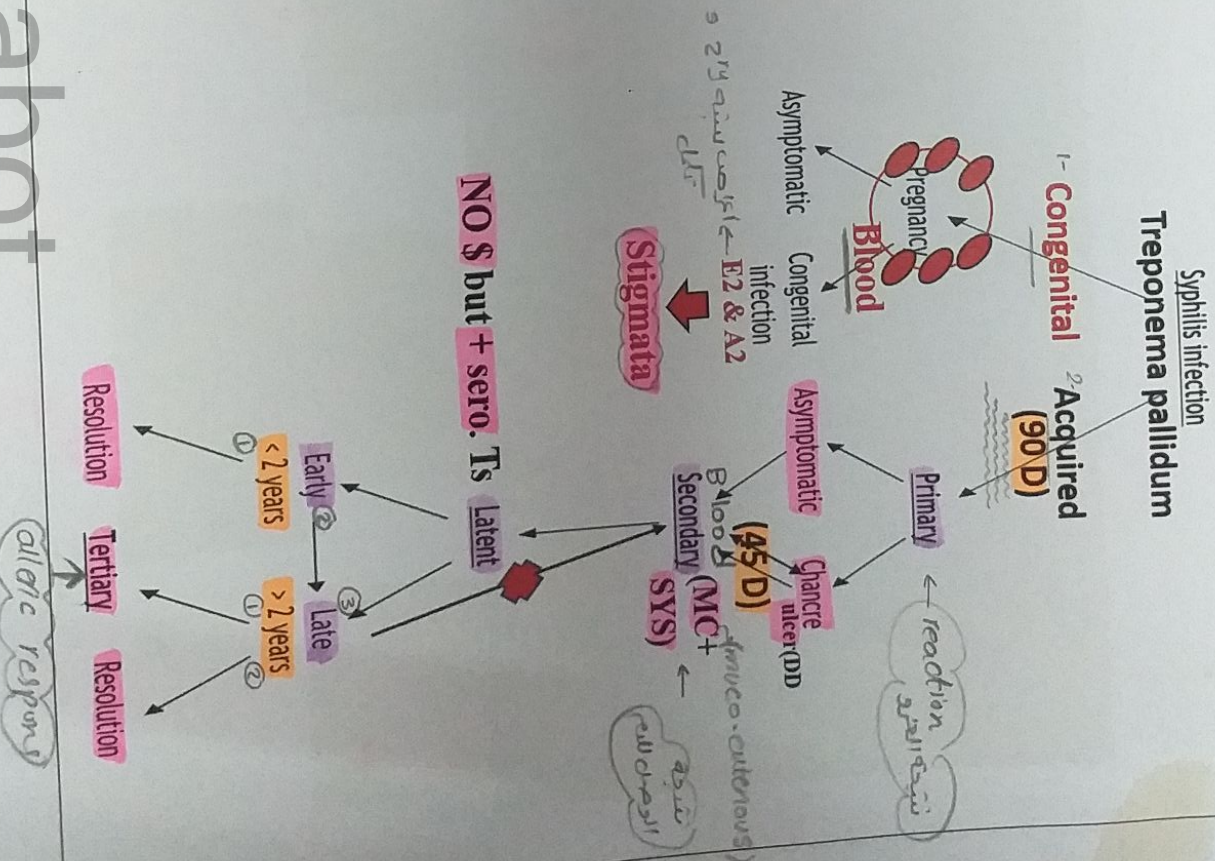
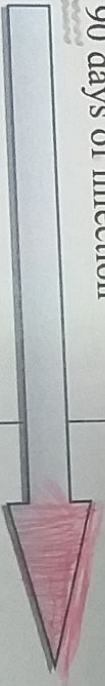
- Vertical transmission: mother baby.

Incubation period: 9 - 90 days of infection

Clinical Picture:

Diagnosis: SPECIFIC – NON-Specific.

Treatment: penicillin



• Organism: *Treponema pallidum* - spiral

No

• Artificial media

• Stain

Killed by:

• Heat

• Dryness

• Washing

• Mode of infection:

Acquired:

• Sexual transmission: **Most common**

• Accidental: direct (doctor), indirect (toilet seats) (very rare)

• Fresh blood transfusion: if the donor is infectious.

• Congenital:

• **Vertical transmission**: mother to baby.

• Incubation period: 9-90 days of infection

• Stages

• Acquired.

• Congenital

Dark Ground Microscopy

(corkscrew rotation motility)

دوران الحلزوني

ACQUIRED SYPHILIS

Primary stage (chancre)

Local reaction → papule → syphilitic ulcer

- Num: Single.
- SITE: genital and extra genital → according to sexual behavior.
- Shape: round or oval
- Edge: sloping
- Floor: clean and fleshy red? → no bleeding as syphilis cause thrombosis, end arteries obliteration.
- Base: Indurated
- Pain: Painless - 2/3 back inf. cause pain also
- LN: same + bilaterally
- Healing: 2 - 6 weeks by thin atrophic scar.

Differential diagnosis

Chancroid - Lymphogranuloma venereum. - Granuloma inguinale. -
Herpes progenerialis - Behcet's disease - Traumatic ulcer - Fixed
drug eruption

Secondary syphilis (MC + Sys)

T. pallidum gain access to blood stream

Skin Rash (great imitator) → Polymorphic → Newer

- **Vesiculates (vesicles)**
 - Bilateral.
 - Symmetrical.
 - Symptomless (non-itchy).
 - palms and soles (characteristic).
- **Hair**
 - Patchy, non-cicatricial alopecia (moth-eaten alopecia).
- **MM**
 - Mucous patches + snail track ulcers
 - Genital condyloma lata.
 - D.D.: Viral condyloma acuminata



DIFFERENCE BETWEEN CONDYLOMA ACCUMINATA AND CONDYLOMA LATA

Human papilloma virus
Condyloma acuminata

2nd stage of syphilis
Condyloma lata

Surface

Rough verrucous

Flat moist

Shape

Pedunculated

Sessile

Base

Soft

Indurated

Bleeding

Bleeds easily

Does not bleed easily → end closure every abnormal

Dark ground examination

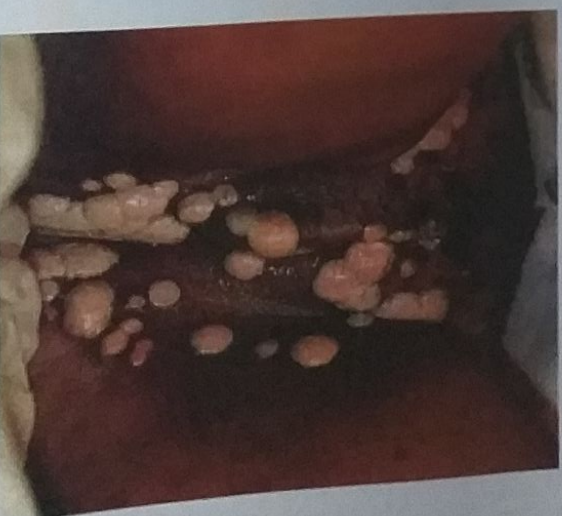
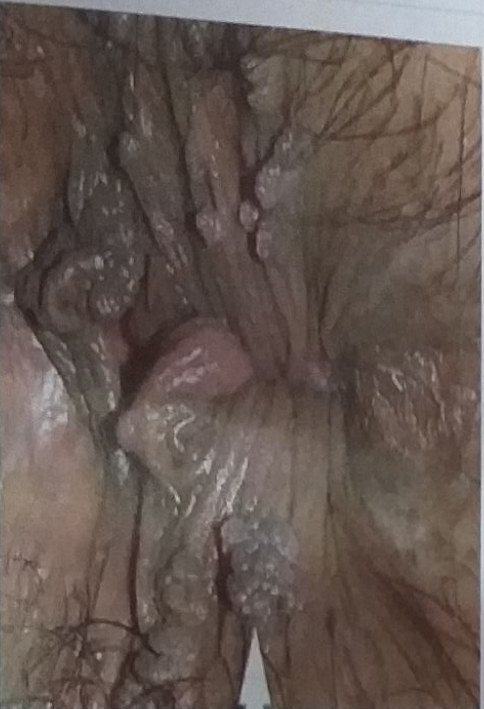
Negative

Positive

Serology for syphilis

Negative

Positive



LATENT SYPHILIS

(NO \$ BUT POSITIVE SEROLOGY)

Spontaneous cure: = resolution

Serological tests: negative and the disease "burnt out" (30% of cases).

Persistent latent Syphilis:

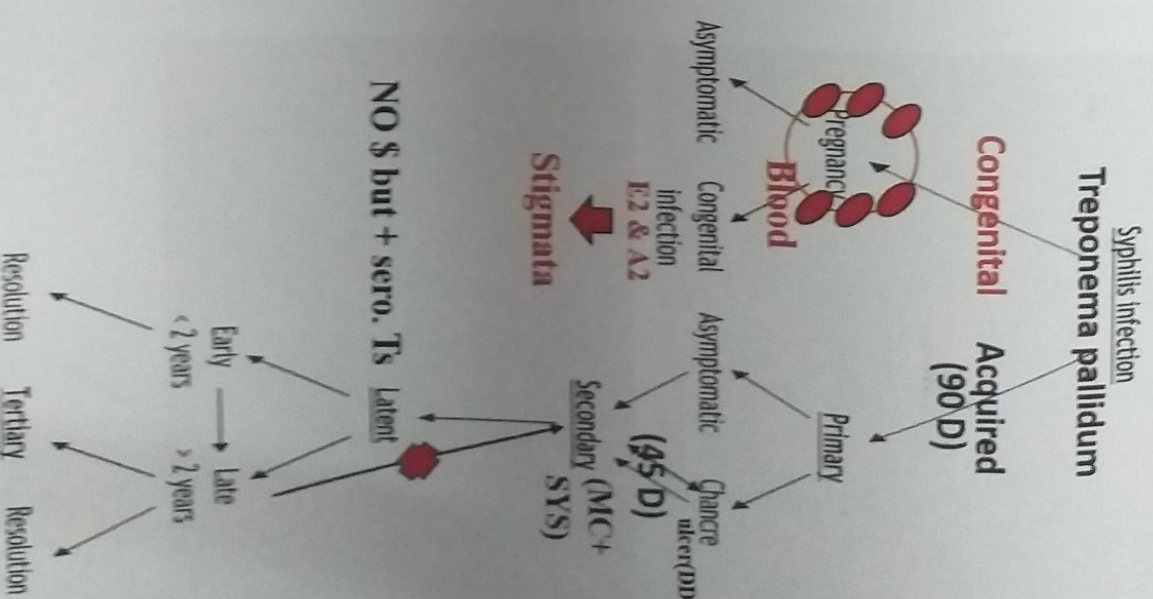
(Serological tests) positive and no manifestations develop (30%).

Benign tertiary Syphilis:

Gumma: in skin, mucous membranes, bones or internal organs (15%).

Chronic syphilitic inflammation of vital organs:

manifestations of cardiovascular and neurosyphilis (25%).



TERTIARY STAGE (GUMMA)

ALLERGIC RESPONSE

- **Etiology:** allergic response to T.P
- **Duration:** 3 - 7 years after chancre.
- **Num:** single or multiple
- **Shape:** polycyclic nodules that ulcerated (gumma)
- **Site:** subcutaneous + Any organs.
- **Edge:** punched out ← (مخاريط الجوزية)
- **Floor:** yellow necrotic material
- **Base:** Indurated
- **Pain:** Painless
- **LN:** NO LN enlargement → as there is no organism.
- **Healing:** atrophic scar.



CONGENITAL SYPHILIS

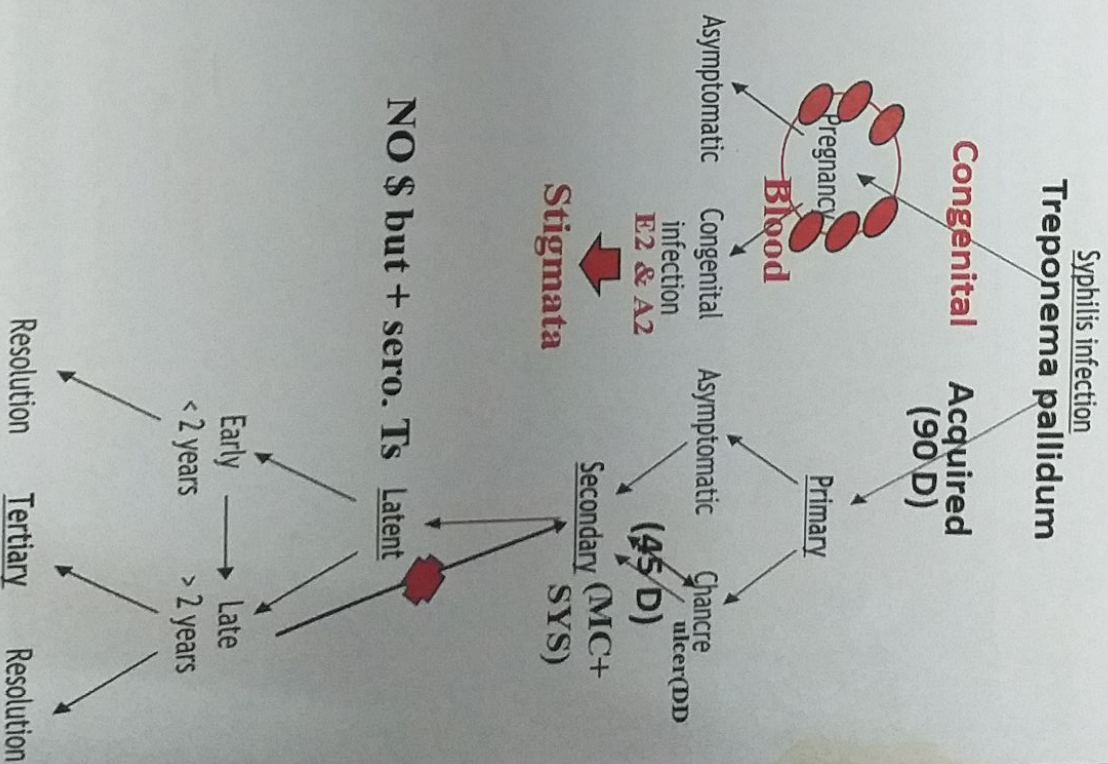
(TT → CROSS PLACENTA → NO 1RY STAGE → NO CHANCERE)
 داء السيليس من المشيمة لا يمر بالمرحلة الأولى

Primary stage:

((Syphilitic placenta is usually bulky, heavy, pale and greasy, infarcts are common?))
 المشيمة السيليسية عادةً تكون كبيرة الحجم، ثقيلة، باهتة ودهنية، تكون النخرات شائعة.

May be:

- Escape infection. / asymptomatic
- Miscarriage (12 - 16th week (placenta formation).)
- Stillborn macerated baby.
- Born with syphilis \$.
- (Born well) develops signs of ECS during 1st 2 years
- (Born well) develops signs of LCS after 2 years.



Early Congenital Syphilis (ECS)

(2ry S + Bone and joints)

- 1st 2 years.
- The skin rash:

mcQ → Vesiculobullous eruptions (never occur in acquired syphilis).

- Rhagades: around MM (fissured) and heal by radiating scars
- Purpuric eruption: thrombocytopenia.

- Bone and joint.

- Rhinitis with highly infectious nasal discharge.

Late congenital syphilis

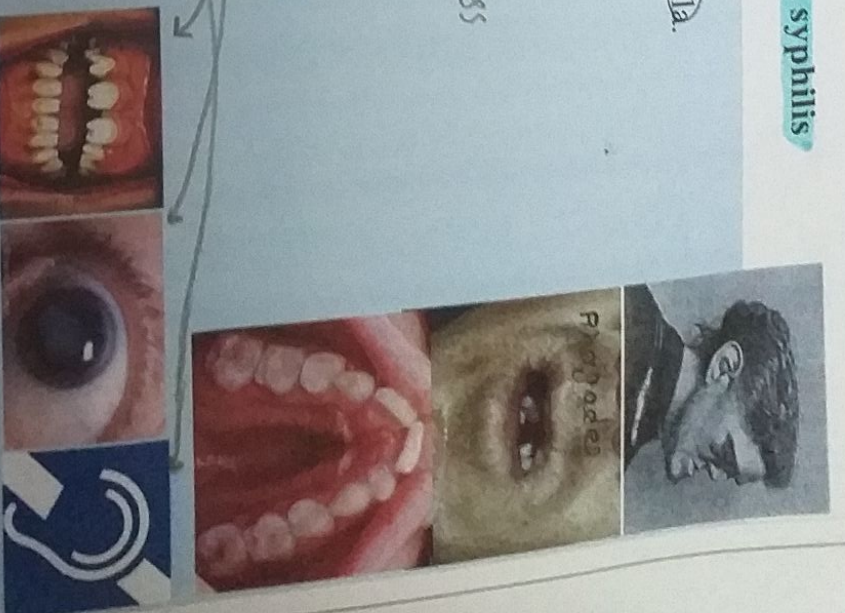
5 years.

Frontal bossae - Saddle nose - Short maxilla.

- Rhagades
- Hutchinson's teeth
- High arched palate.
- Sabre tibia (فك عظمي مشوكة / فك عظمي مشوكة)
- Clutton's joint. → synovitis → painless
- Paroxysmal cold hemoglobinuria

Hutchinson's triad (pathognomonic):

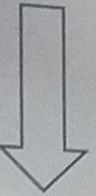
- Hutchinson's teeth
- Interstitial keratitis
- Eighth nerve deafness



Stigmata

Congenital Syphilis

(permanent marks)



- ✓ Syphilitic facies: frontal bossing, saddle-shaped nose, prominent mandible and short maxilla.
- ✓ Corneal opacities after interstitial keratitis.
- ✓ Hutchinson's teeth.
- ✓ Rhagades: radiating scars peri-nasal, perioral and perianal
- ✓ Sabre tibia.

Diagnosis of Syphilis:

1. Dark field microscopy: (primary & secondary stages)

2. Serology:

A. Nonspecific antibodies:

Standard Tests of Syphilis: antibody titre correlates with the disease activity

- Low Specificity → false positive ← *metagenallergic*
- High Sensitivity → Screen - FU therapy.
- VDRL & RPR

B. Specific antibodies:

Antibody titre correlates poorly with the disease activity

Fluorescent Treponemal Antibody absorption (FTA-ABS IgM) test:

- First specific test to become positive
- IgM → Not cross placenta → can diagnose neonatal syphilis

Treponema Pallidum Immobilization (TPI) test: Most specific

Treponema Pallidum haemagglutination (TPHA) test: Midway

3. C.S.F: (neurosyphilis)

Treatment of Syphilis:

Benz. Penicillin G

If success ttt → ↓ titre

1. Penicillin: (Long acting benz. penicillin)

- Dose: 1.2 million U/twice weekly
- Total dose: 12 Million U
- S/E: *massive killing of organism*

Herxheimer reaction (therapeutic shock): 1st dose: → sudden release Ag → treated with steroids ← *Penicillin dose*

- Therapeutic paradox: long term ttt → fibrosis
- Penicillin allergy

2. Tetracyclines 2 gms daily for 15 days
3. Erythromycin 2gms daily for 15 days

Early Syphilis:

Late Syphilis

Benzathine penicillin G	2.4 million U Single Dose	2.4 million units/W Up to total dose
Erythromycin	500 mg / 4 times daily 15 days.	500 mg / 4 times daily 30 days
Tetracycline		

Congenital Syphilis

A. Prophylactic: ttt pregnant

B. Curative:

Early → penicillin 400,000 u/kg (total Dose) divided over 15 days
Late → as acquired syphilis

OTHER SEXUALLY TRANSMITTED DISEASES

LM4

LYMPHOGRANULOMA VENEREUM

Chlamydia Trachomatis
L-1, 2, 3.

Incubation period

2 - 3 weeks

Organism

Clinical Picture

Multiple adenitis:

- matted and suppurate
- multiple fused abscesses (buboes).
- multiple sinus formation.



Chancroid
croi → cry → pain

Hemophilus ducreyi
gram-negative bacilli

Multiple Papules → pustular → ulcerated

- Pain: tender and quite painful.
- Edges: ragged and undermined
- Base: granulation tissue that bleeds readily on manipulation.
- LN: unilateral + painful + matted.

D.O :- syphilis ← ulcerations



Granuloma Inguinale (Kalemat)

Calymatobacterium granulomatis

Try: Papules or nodules → ulcerate

- LN: No → Granuloma mimicking lymph nodes (pseudo-buboes).

Complications:

- Stenosis of the urethral, vaginal or anal orifices,
- massive elephantoid oedema
- malignancy.



Chancere**Incubation period**

9 - 90 days

Number of lesions

single

Pain

painless

Edge

sloping

Base

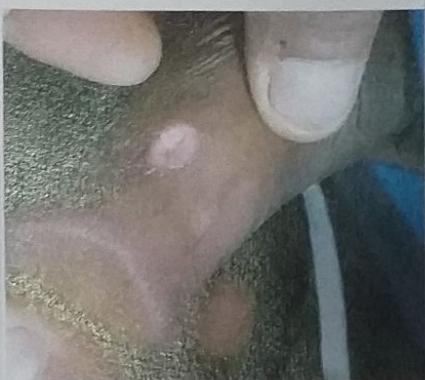
firm indurated

Bleeding

does not bleed easily

Lymph nodes

bilateral, painless, discrete, firm, never suppurate

**Chancroid**

4-10 days

multiple

painful

ragged undermined → deep ulcers

soft

bleeds easily

tender, matted, suppurate and form sinuses



14

ACQUIRED IMMUNODEFICIENCY SYNDROME



You TubeTM
SCAN TO WATCH

ACQUIRED IMMUNODEFICIENCY SYNDROME END STAGE OF HUMAN IMMUNODEFICIENCY VIRUS (HIV)

present in Africa, less transmission

Organism: HIV1 & HIV2 → Retrovirus.

Transmission:

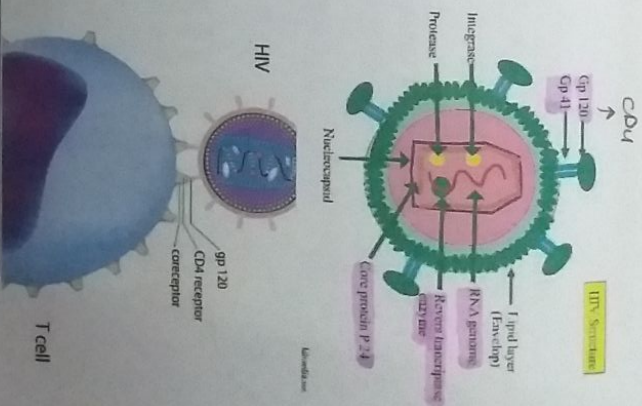
- Highest Conc. in blood and genital secretions.
- Transmission:
 - ✓ Sexual
 - ✓ Mother to child: placenta, during delivery (M/C) or breast milk.
 - ✓ Needles by IV drug abusers
 - ✓ Accidental needle
 - ✓ Blood

NB: No transmission by Mosquitos bites

Pathogenesis:

- HIV affect CD4 + T lymphocyte causing decrease number & function → immune dysfunction.
- AIDS cause opportunistic infections and neoplastic processes.
- Internal infections are the major cause of death.

Two strands of RNA and use a DNA polymerase enzyme called reverse transcriptase to convert viral RNA to DNA



Clinical picture:

IP: 3 to 6 weeks

Group1: The initial HIV infection (Acute HIV illness) Seroconversion:

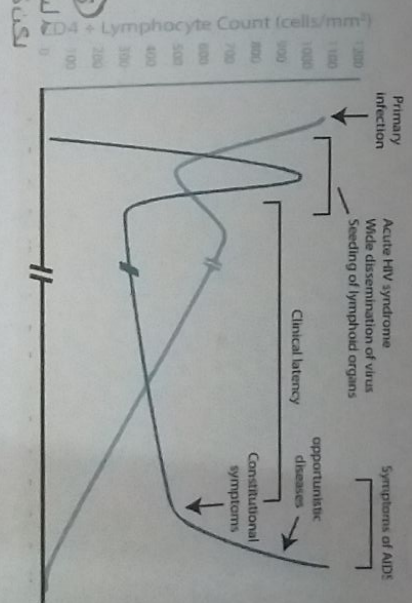
- Acute mononucleosis-like syndrome: 50% → fever, — (10-14 days)
- Seroconversion:
 - Transient rise (Viral load) + antigen (glycoprotein) + (dropped CD4) +
 - No detectable (Anti-Body) → forms within 3 months

Group2: Asymptomatic HIV infection (clinical latency):

- Asymptomatic
- Detected Anti-body against glycoprotein
- No detectable Viral load + CD4 > 350 + antigen (glycoprotein).

Group3: Symptomatic HIV infection (chronic generalized lymphadenopathy).

Group4: Progression to AIDS.



Complications: Progression to AIDS

((Opportunistic infections + Malignancy))

1. Prolonged constitutional symptoms:

- Fever - night sweats
- weight loss, and diarrhea.
- chronic fatigue

2. Opportunistic infections:

mcd Pneumocystis carinii pneumonia (most

serious)

- oral candidiasis
- oral hairy leukoplakia: *EBV*
- herpes simplex/zoster
- fungal infections

3. Malignancy:

mcd → Kaposi's sarcoma: *HHV8*

- Lymphomas

Diagnosis:

1. Detection of viral antibodies in the serum:

- ELISA (sensitive). *initial screening*
- Western blot (confirmatory). *definitive*

2. Detection of viral antigens in blood:

- P24 → (core protein)

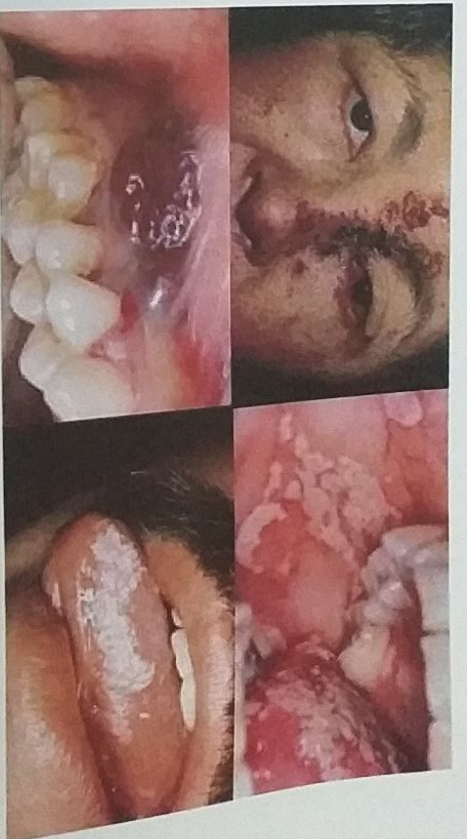
3. PCR:

- Detection of viral RNA in blood
- Reverse transcriptase

- high: Acute disease and late in AIDS stage.

Measures of CD4 + T lymphocytes

- clinical progression and therapeutic response.



Management:

(No curative treatment for HIV)

- Prevention
- Encourage casual social life contact with known HIV-infected persons.

Antiretroviral therapy:

- CD4 + T-lymphocyte counts of less than 500/microliter.

Antimicrobial prophylaxis:

- Pneumocystis carinii CD4 + T-lymphocyte counts of less than 200/microliter

15

MALE INFERTILITY

CONTENTS LIST

Testis (Anatomy - Histology - Function)
Evaluation of male infertility
Treatment



CHAPTER 15

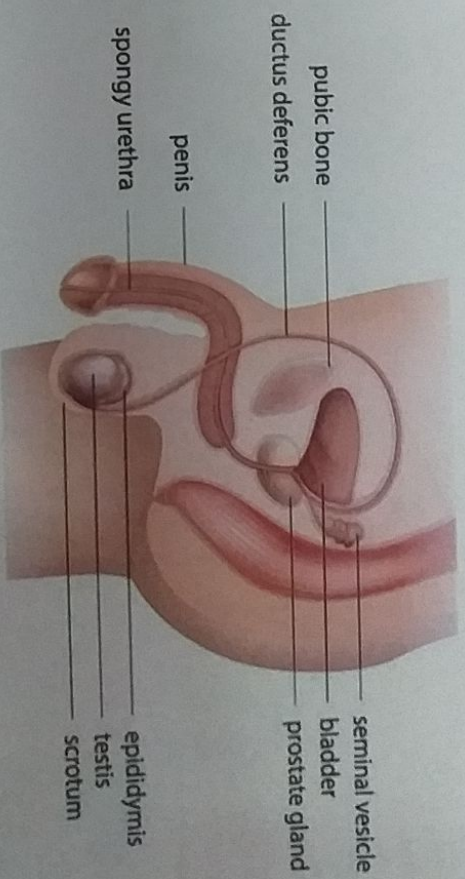
MALE INFERTILITY

➤ Fertility: male genital tract

- Testis (testosterone – sperm)
- Epididymis (Maturation) (sperm)
- Vas (Transport)
- Seminal vesicles (Nutrition)
- Ejaculatory duct
- Prostate (liquify semen)
- Cowper G – Littre G (lubrication-PH)

➤ Sexology(penis):

- Erection
- Ejaculation



FERTILITY

TESTIS (ANATOMY - HISTOLOGY - FUNCTION)

Anatomy (Out → In):

Testis: 2
Each one:

- Skin: no fat
- Layers 3
- Tubes 250 - 300
- Each lobe: 2 - 3 seminiferous T.
- Each seminiferous T: 50 cm
- Total length: 500 meters

Histology:

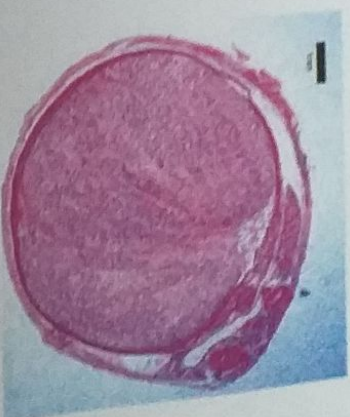
1. Seminiferous tubules.
2. Interstitial tissue.

1. The seminiferous tubules:

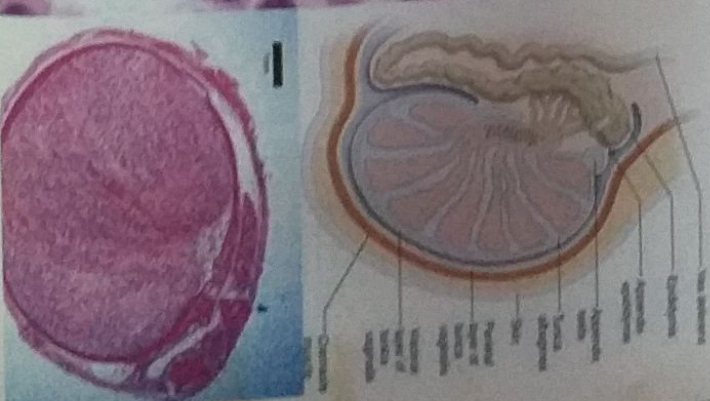
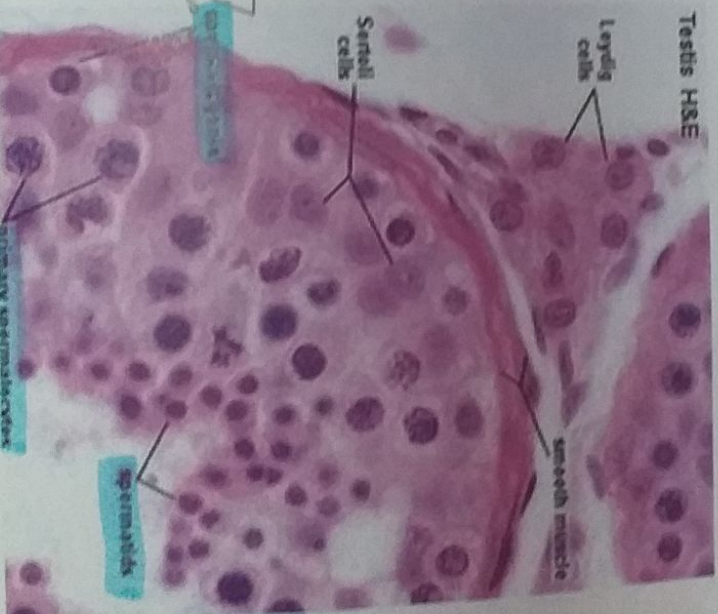
- A. The germ cells (Under FSH) → (cells in tubules)
- B. The germ cells = spermatogonia

The Sertoli cell (Under FSH):

1. Non secretory F (Like Mother):
 - Formation of blood-testicular barrier: Toxic substances & Sperm. → *dead ones*
 - Phagocytosis & nutrients to the germ cells.
 - Mechanical support of germinal epithelium.
2. Secretory function: (Embryo - Adult)
 - Androgen-binding protein (ABP) → *androgen binding protein*
 - Inhibin → (-) to FSH → *block luteal stop sperm cells*
 - Mullerian Inhibitory Factor (Fetal Life)
 - Transferrin (Marker)
 - Growth factors



Testis H&E



The germ cells:

- Spermatogonia (2N)
- **primary spermatocytes (4N)** (*largest cell*)
- secondary spermatocytes (2N)
- spermatids (1N)
- Sperm (spermatozoa) (1N)

NB:

A. Sperm production (75 D): 3 Stages:

1. **Spermatogenesis:** Division spermatogonia (2N) → Spermatid (1N)
2. **Spermiogenesis:** change Spermatids → sperm.
3. **Spermiation:** Release sperms to epididymis

B. (90 days) = Sperm production (75 D) + Maturation in Epididymis (15 D) → 3 months needed for TTT and Re-evaluation

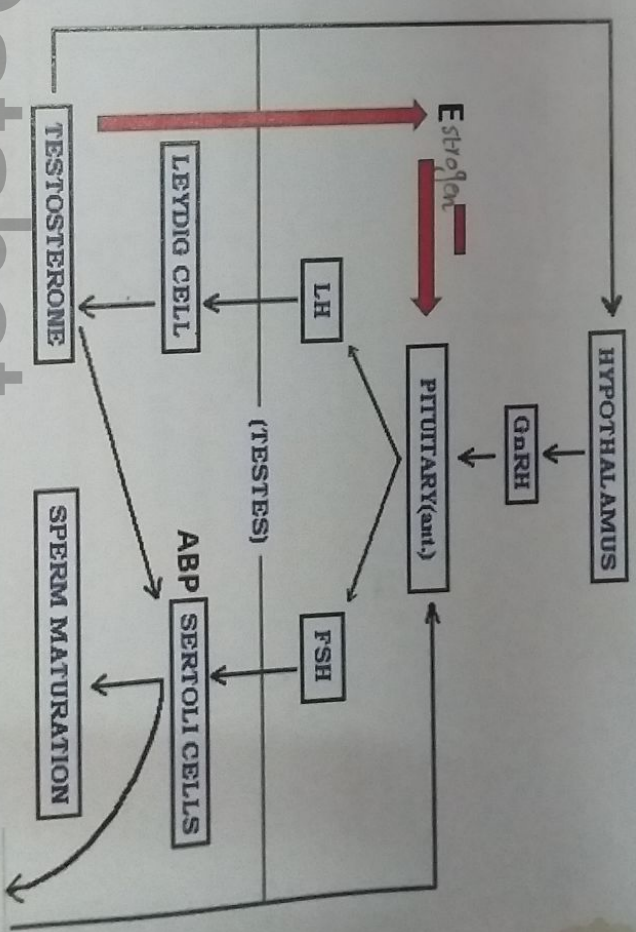
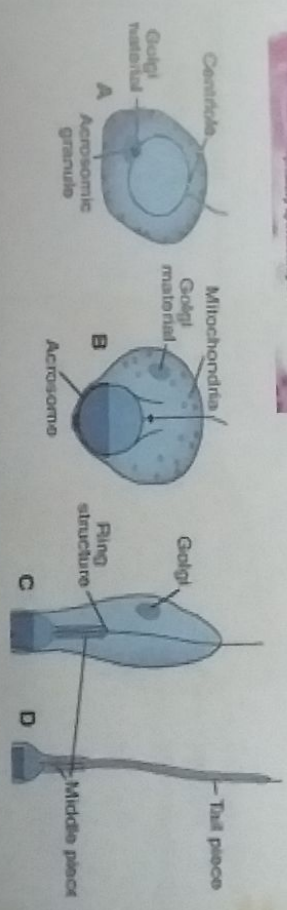
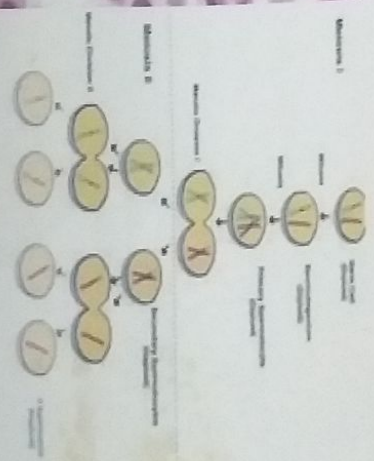
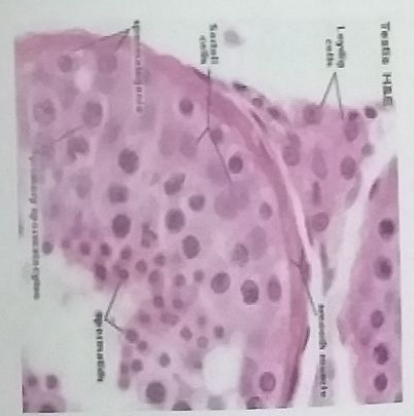
2. Interstitial tissue:

A. Leydig cell (under LH):

- Androgens (mainly testosterone).
- Testosterone converted to dihydrotestosterone by 5-α reductase → (secondary sexual Character)

B. Others: areolar tissue, collagen fibers, elastic fibers, blood vessels, and lymphatic vessels

Seminal fluid (95%)			Sperm (5%)
Seminal vesicles (60%)	Prostate (30%)	Cowper's + Littre's G (prosemen) (5%)	Testis + Epididymis (5%)
Alkaline	Acidic	Neutralization	



Evaluation of male infertility

History:

History of infertility:

- Duration - Previous pregnancies
- Evaluation of the partner
- History of gonadotoxins, smoking, alcohol, marijuana, and cocaine.

Sexual history:

- Erectile dysfunction (ED). Spermicidal lubricants.
- Timing and frequency of intercourse?

- Premature ejaculation - (Retrograde ejaculation) *Bladder if retro*

- Loss of libido *undescended testes*

Medical history: puberty onset - cryptorchidism - mumps - GHI - Pituitary adenoma - DM

Drug history - *chemotherapy*

Surgical history: bladder neck operations - retroperitoneal LN removal (testicular cancer) → absent - retrograde ejaculation

Family history: chromosomal

Clinical examination:

General examination:

- Gynecomastia. *↑ estrogen ↑ prolactin*
- Body hair distribution - muscular mass - skeletal portion

Local examination:

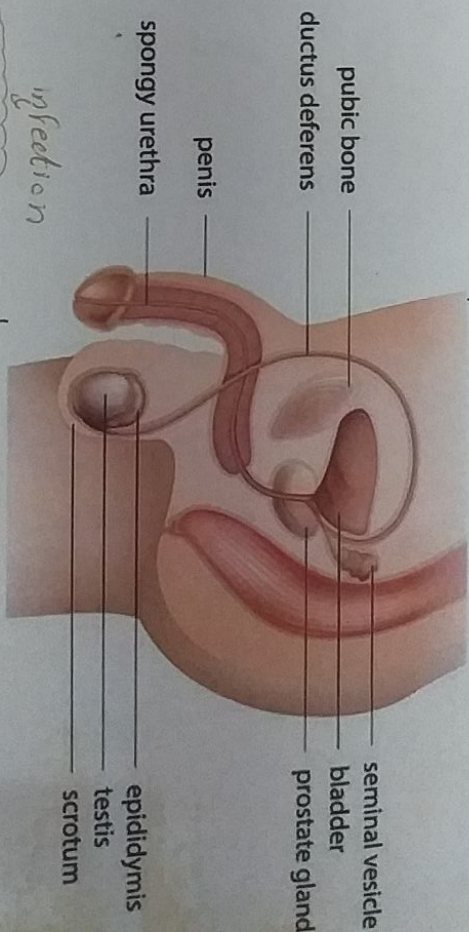
- Penis.
- Examination of testes.
- Examination of the epididymis.
- Examination of the spermatic cord.

neck of scrotum - i. Vas deferens may be absent
the bladder ii. Detection of varicocele

P/R: = Per Rectum

- Prostate examination → Prostatic smear → microscope → more (10 pus C/HPF) → prostatitis
- Seminal vesicles are normally not felt (felt obstruction or infection)

check 12 weeks



Zoosperm = sperm / spermia = semen

Investigations:

A. Semen analysis:

Volume: 2-6 ml after 3-5 days of sexual abstinence ^{ليل}

- Aspermia: No semen, e.g. retrograde ejaculation.
- Hypo - or oligospermia: low semen volume < 1.8 ml. (e.g. bilateral cong. absent vas, ejaculatory duct obstruction)
- Hyper - or polyspermia: > 6 ml

PH: 7.2 - 8 (alkaline)

Liquefaction: within 10 - 20 minutes

Sperm count: > 20 million/ml (normozoospermia):

- Azoospermia: No spermatozoa in semen (obstructive or functional)
- Oligozoospermia: sperm < 20 million/ml
- Polyoospermia: > 250 million/ml

Percent of motile sperm: > 60% in the first hour

Forward progressive motility

- Asthenozoospermia: decreased motility of sperm.

Abnormal morphology < 30%

- Teratozoospermia: > 35% abnormal spermatozoa
- Globozoospermia: round heads of sperm (no acrosomes)

Percent of viable sperm: > 50%

Seminal vesicle

- Necrozoospermia: All sperm are dead. (Eosin-Negrosin) ^{الحيات لا تتحرك}

Dead is dead

Fructose content: 1200 µg/ml

Pus cells 3 - 5/High power field

- Pyospermia: > 6 pus cells/high power field

B. Hormonal:

- FSH, LH
- Testosterone
- Prolactin
- thyroid hormonal assessment

Azoospermia

Obstructive:

- ✓ - Epididymal obstruction.
- ✓ - Vasal obstruction. ^{seminal vesicles}
- ✓ - Ejaculatory ducts obstruction.

Functional:

- ✓ - Congenital: cryptorchidism.
- ✓ - Chromosomal: Klinefelter syndrome
- ✓ - Traumatic.
- ✓ - Hormonal.
- ✓ - Inflammatory.
- ✓ - Neoplastic.
- ✓ - Radiation and chemotherapy

How to differentiate between function and obstructive azoospermia?

C. Testicular biopsy (Differentiate):

Functional azoospermia:

- Sertoli cell only S. ← (الخلايا فقط)
- spermatogenic arrest ← (الخلايا متوقفة)

Obstructive azoospermia

Treatment:

- ✓ Medical, e.g. hormonal.
- ✓ Surgery for correction of obstruction, varicocele ligation
- ✓ Assisted reproductive techniques (ART):
 - In vitro fertilization (IVF) ← (البويضات)
 - intracytoplasmic sperm injection (ICSI). ^{الخلايا}

16

SEXOLOGY

CONTENTS LIST

Anatomy of the penis
Mechanism of penile erection
Neurophysiology and neuroanatomy
ERECTILE DYSFUNCTION

SCAN TO WATCH
You Tube™



CHAPTER 16 SEXOLOGY

Sexology (Anatomy of penis):

Erection → erectile Dysfunction.
Ejaculation → Premature Ejaculation.

Anatomy of the penis

The body of penis (3 corporeal bodies):

- Two corpora cavernosa: sinusoidal spaces separated by trabeculae
- Single corpus spongiosum urethra & glans penis.

The root of penis consists of:

- Two crura:
 - Two corpora cavernosa extension (attach to ischiopubic ramus)
 - Covered by ischioavernosus muscles
- The bulb:
 - Corpus spongiosum extension.
 - covered by the bulbocavernosus muscle

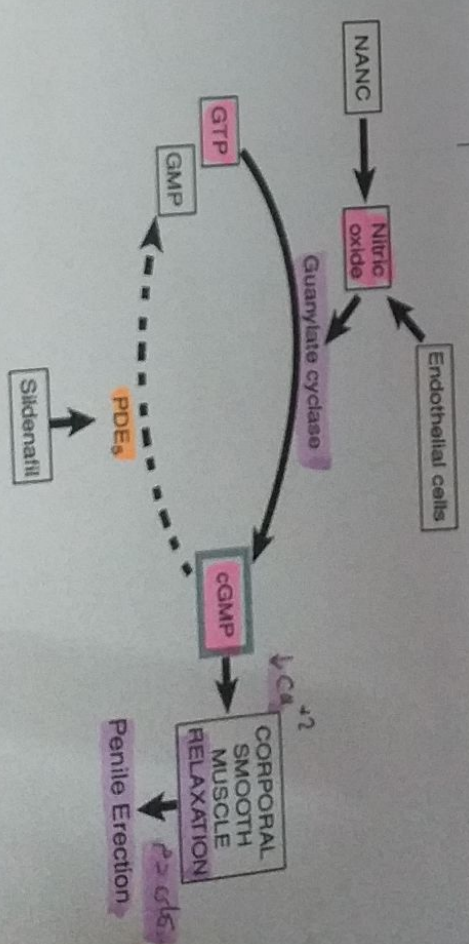
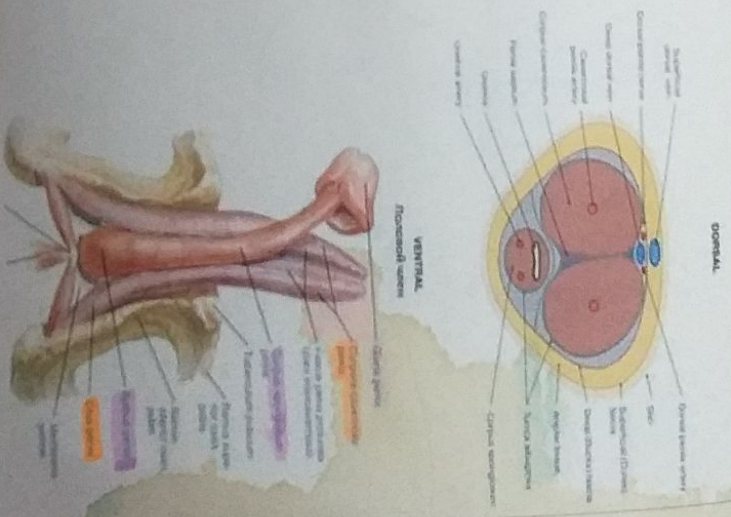
Mechanism of penile erection

Systems:

- Normal psychological status ✓
- Adequate hemodynamics (A&V) ✓
- Normal endocrinological status ✓
- Normal neurogenic pathways ← mda

Local:

Erection: Nitric Oxid > high cGMP > VD. →
Loss of Erection: PDE 5 degrade cGMP
phosphodiesterase enzyme 5



NEUROPHYSIOLOGY AND NEUROANATOMY

Autonomic and Somatic nerve fibers

Autonomic fibers:

1. Sympathetic pathway:

- Origin: Thoracolumbar T11 to L2
- Neurotransmitter: norepinephrine
- Action: vasoconstriction
- State: flaccid = anti-erection

2. Parasympathetic:

- Origin: Sacral (S2 to S4).
- Neurotransmitter: acetyl choline → NO → High cGMP
- Action: Vasodilation
- State: erection

NB: nonadrenergic-noncholinergic neurotransmitters. e.g. VIP, NP-Y and substance P

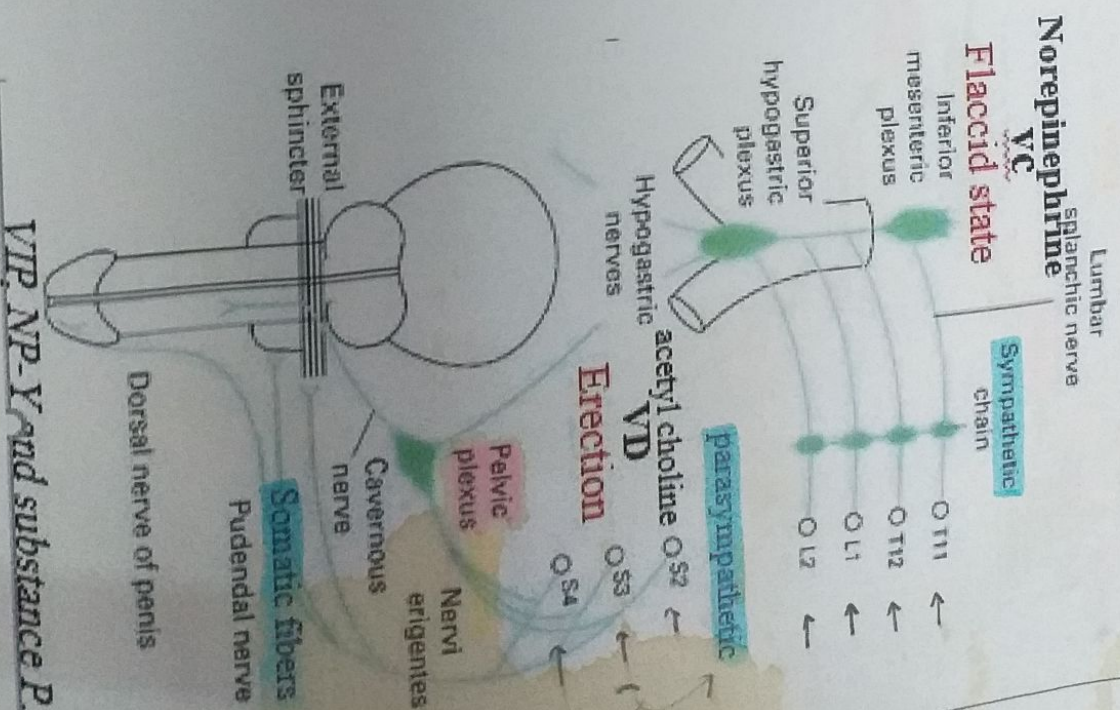
Somatic fibers (pudendal nerve) (S2-S4).

1. Motor: TO

- Ischiocavernosus
- Bulbocavernosus muscles.

2. Sensory: From

- glans penis & penile shaft (Sacral Plexus)





ERECTILE DYSFUNCTION

INABILITY TO ACHIEVE OR MAINTAIN ERECTION SUFFICIENT FOR VAGINAL PENETRATION

Etiology:

Psychogenic: most common

Organic:

- Intra- or interpersonal factors (Normal NPT)
- Vasculogenic (arterial and/or venous).
- Neurogenic.
- Endocrinal.
- Mixed. e.g.: D.M.

History:

Personal history: Marital – Occupation - Personal satisfaction - Special habits - Sleep disorders.

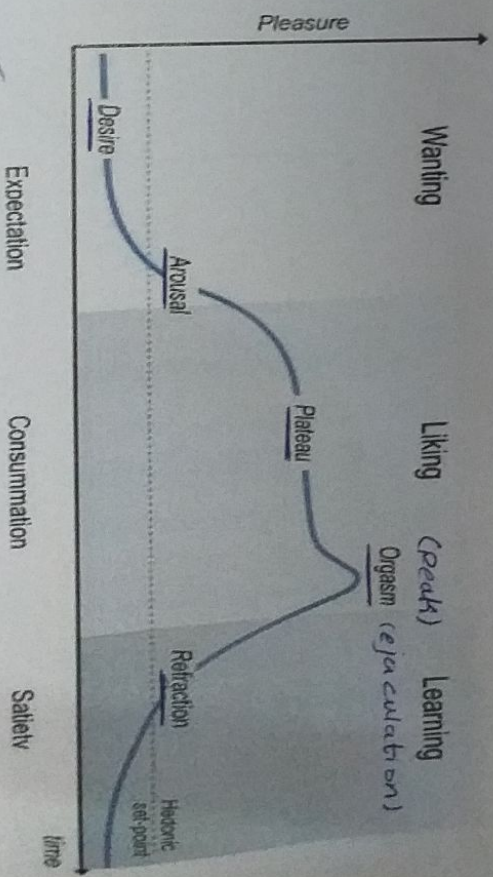
Sexual history: Sexual development- Sex education- Depression- anxiety, or phobias.

History of ED:

- Onset, course, duration.
- Desire: low – normal – high.
- Sexual arousal
- Erection: In the morning, during masturbation.
- Ejaculation: premature – normal – retarded
- Orgasm: present – absent

Medical and surgical history

History about the wife/partner.



Clinical examination

General examinations:

- **Endocrine:** Secondary sex characteristics - breast for swelling. *↑ prolactin*
- **Vascular:** Femoral and lower extremity - Temperature *↓* trophic changes. *↑ atherosclerosis*
- **Neurological examination:** diabetic P - neurodegenerative disorders *↑ peripheral neuropathy*
- Signs of systemic disease that may lead to ED.

Genital examination:

- **Penis:** size - Peyronie's plaque - balanitis - urethritis. *↑ erection pain*
- **Testes:** - small - atrophic → *↓ desire* & *↓ arousal*
- Digital rectal examination (P/R)
- Testing the genital and perineal sensations.

Investigation

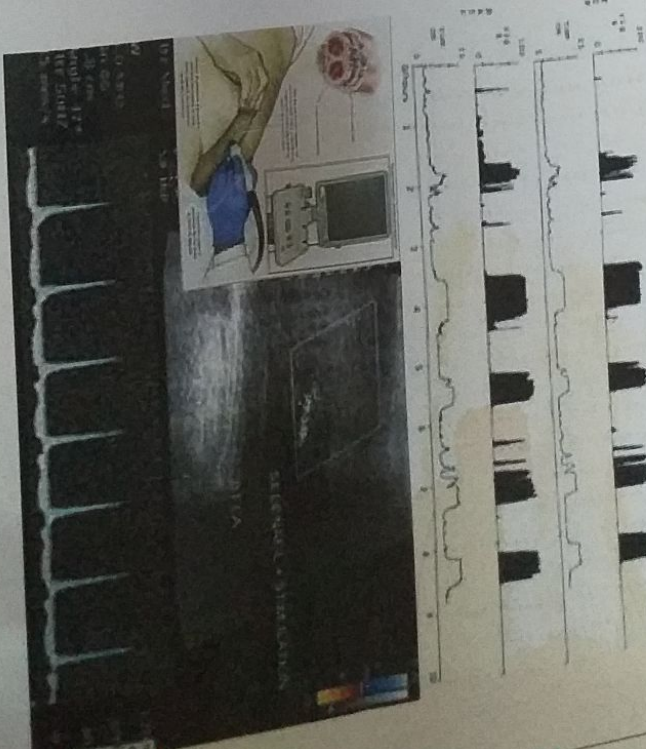
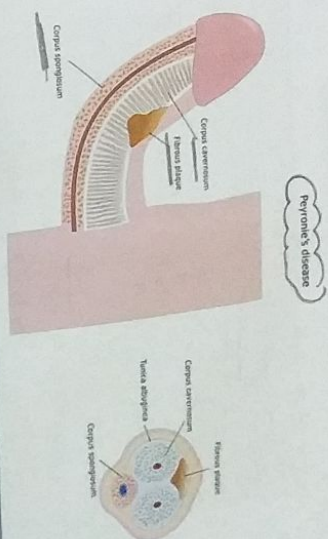
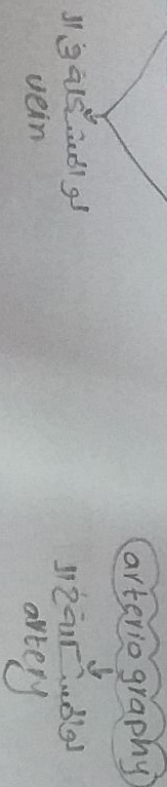
Rigiscan (sleep): detect

- Circumferential *(قاس محيط)*
- Rigidity of the penis during. *(الصلابة)*
- Duration *(المدة)*
- Normal: psychogenic ED.

Evaluation of penile arteries:

- These include Doppler, penile duplex U/S. *← patent blood flow*
- injection of vasoactive substances (ICI). *↑ arterial erection*

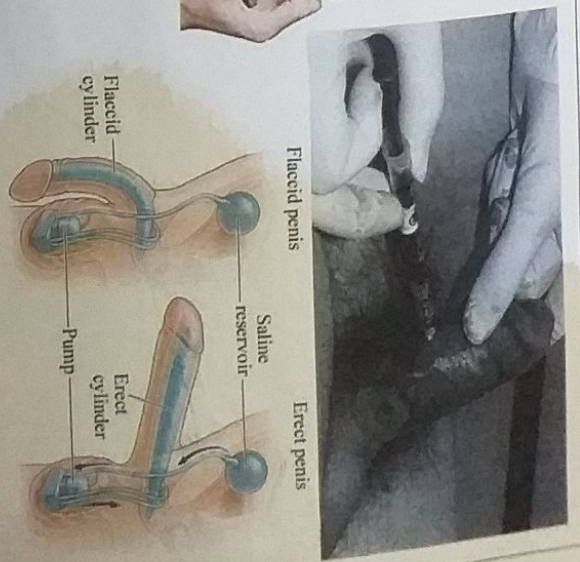
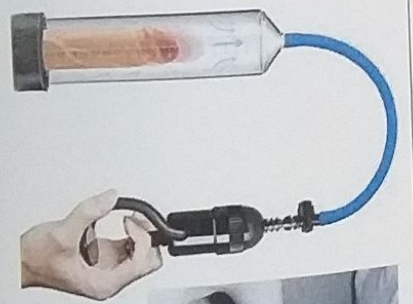
Cavernosometry and cavernosography.



↑↑↑↑↑↑↑↑↑↑
cAMP

Treatment

- PDE5 inhibitors: Sildenafil citrate, Tadalafil, Vardenafil.
- Intracorporeal injection (ICI):
 - Papaverine hydrochloride
 - Phentolamine
 - Prostaglandin E-1.
- Hormones: bromocriptine (in hyperprolactinemia)
- Vacuum device. -ve
- Surgical: Penile implants (penile prosthesis).



PREMATURE EJACULATION

The inability of the male to control ejaculation long enough to satisfy his partner in at least 50% of their sexual encounters, when there is no female orgasmic dysfunction

Etiology

A. psychogenic in origin.

- Mal-conditioning: (تكوين عادات)
- Fear of being discovered.
- Prostitute. (عزلة)
- Masturbation.
- Anxiety.

B. Organic causes (rare).

- Chronic prostatitis, and multiple sclerosis

Treatment

- Reassurance.
- Drugs: Thioridazine - clomipramine and SSRI.
- Sex therapy: squeeze technique.
- Sensation: local anesthetics or condoms.